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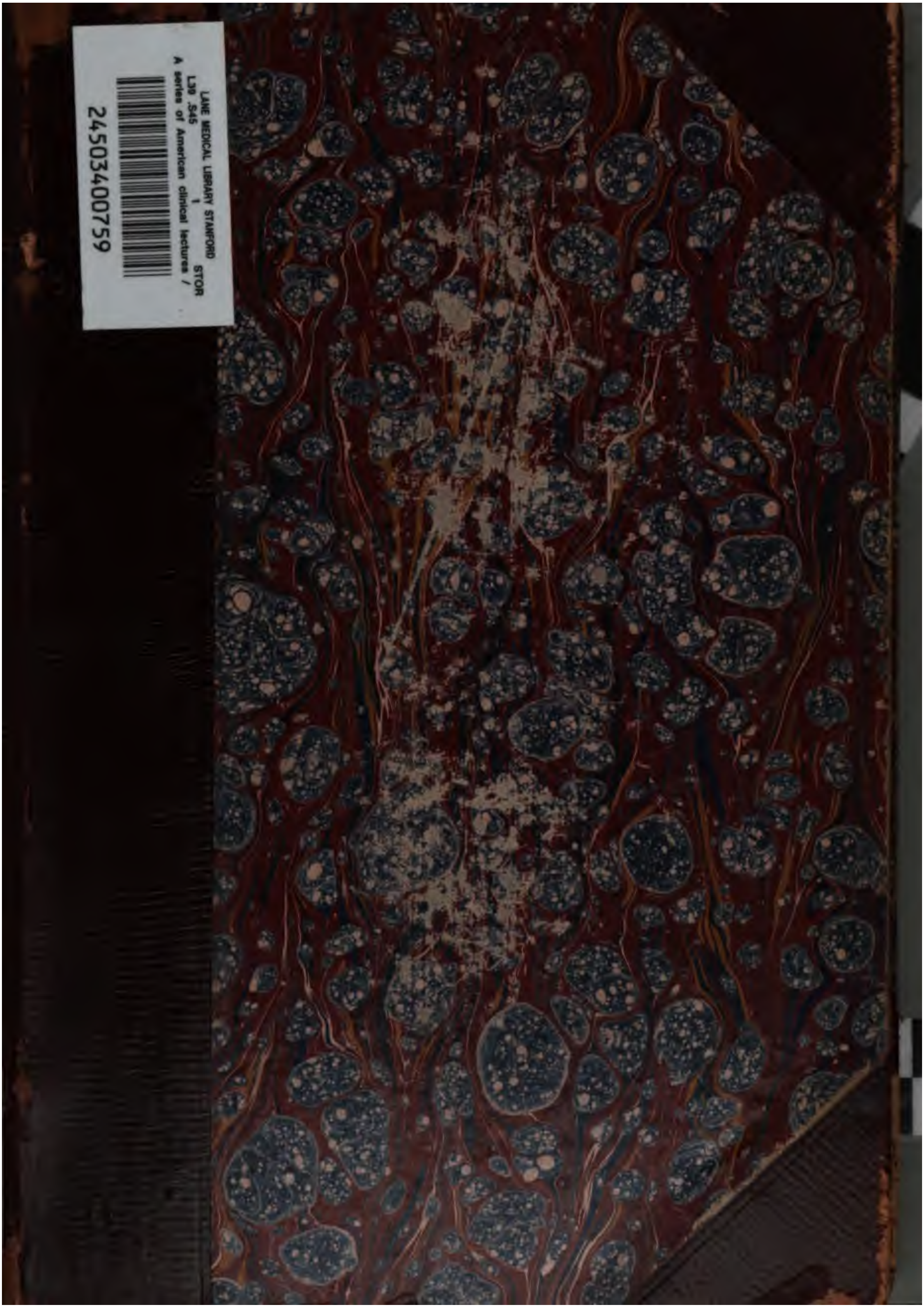

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A SERIES OF

AMERICAN CLINICAL LECTURES

EDITED BY

E. C. SEGUIN, M.D.

Volume I.

JANUARY—DECEMBER, 1875.

NEW YORK:
G. P. PUTNAM'S SONS,
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Number 1. **January, 1875**

ON

HIP-JOINT DISEASE.

BY

LEWIS A. SAYRE, M. D.,*

Professor of Orthopœdic and Clinical Surgery in Bellevue Hospital Medical College, New York.

Gentlemen :—Among the numerous cases present at our clinic to-day, we find three of Hip Disease, and, strange to say, they are typical representations of this disease in each of its three different stages. We shall, therefore, avail ourselves of this opportunity to make some extended remarks upon the subject.

Hip Disease, or *Morbus Coxarius*, as it is ordinarily called, is more frequently observed in childhood than at any other period of life, although I have frequently seen it in the adult. The causes of the disease, judging from my own personal experience, are at most always traumatic. It generally results from an injury, blow, fall, wrench, strain, over-exertion, jump, violent exercise, and sudden checking of the perspiration, or some other violence done to the part, and may occur in any child in the world. In fact, from careful observation of some hundreds of cases, I have found it more frequently in the active, robust, healthy child, than in the dull and sickly one, for the reason that these children are more reckless, wild, and romping, and therefore run more risk of injury than the feeble, sickly child. I know, gentlemen, that this is not the doctrine that you find in any of your text-books, and is, at the present

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*** Delivered in Bellevue Hospital Medical College, December 23, 1874. Phonographically reported by W. S. George.**

time, not generally believed by the mass of the profession. I was taught, and all your standard works and text-books teach you, that it is a disease essentially of a strumous origin, dependent entirely upon a tuberculous or scrofulous constitution. One of the most distinguished writers on Surgery states distinctly, "No struma, no hip disease."

I do not pretend to assert, as some have stated, that a strumous constitution is a prevention of hip disease, but, on the contrary, a delicate constitution, or a child with a strumous diathesis, would be much more likely to have the disease developed, from the same exciting cause, than would the healthy vigorous child. One of these children will be just as likely (or more, perhaps) to be injured by falling from a window, or an apple-tree, or in a railroad collision, as the healthy child ; but these sickly, delicate, strumous children do not climb apple-trees, jump over stone walls, or indulge in any of the dangerous sports of the healthy child, and, consequently, are not so liable to these accidents.

The disease may occur in early infancy, through the carelessness of the nurse allowing the child to fall from her arms or roll from the crib, or in various ways to injure itself. Ordinarily, at this period of life, the disease is not developed, from the fact that the mass of children are tenderly cared for and these accidents avoided; but, from two to ten years of age, when they are large enough to run about and exercise without being under the control of a more prudent person, and before they have arrived at an age of sufficiently mature judgment to guard themselves, is the period of its most frequent occurrence—during the age of recklessness.

The injuries that produce this disease are frequently so slight as, at the time, to pass unheeded by the thoughtless child. As soon as the momentary pain caused by the accident has faded away, the child resumes his games and romps, and neglects to call attention to the slight fall, or wrench, or strain, or bruise which he has received, not being old enough to comprehend its importance.

Later in life, these trivial injuries attract attention and are immediately cared for, which is the reason, I presume, that the disease is not more frequently found in adult life.

The pathological changes that these causes produce are either *Synovitis*, from a rapid and excessive change in temperature; or violent wrenches or strains, tearing from its attachments the ligamentum teres, thereby inducing necrosis of the head of the femur from rupture of the blood-vessel which supplies it; or concussions, falls, jumps, blows, will produce an extravasation of blood in the articular lamella, which sets up an inflammation, the cartilages soon die on account of their low vitality, become eroded and necrotic, interstitial absorption of the bones takes place, due to the constant pressure from muscular contraction, and, finally, exfoliation. But, no matter what the cause, or which of the tissues originally involved, they all, sooner or later, become commingled and included in the general destruction. I don't believe the disease ever commences in the cartilage, as it is entirely without blood-vessels and nerves, and lives simply by imbibition.

For convenience of description, we will divide the disease into three distinct stages, as each represents a different pathological condition of the parts involved, and as the symptoms also vary to a greater or less degree :—

1. The stage of irritation.
2. The stage of effusion.
3. The stage of rupture of the capsule, or perforation of the acetabulum.

In the first stage, or stage of irritation, as it may be called, before effusion has occurred within the joint, the symptoms are not well pronounced, and it often requires a very careful investigation in order to diagnosticate the disease. Generally, the first thing noticed is that the child appears very slightly lame when he first gets out of bed in the morning, or when first he moves about after some hours of rest. This limping or halting gait is so slight as hardly to be observed, and, after a few minutes of exercise, seems to disappear altogether until the following day, or after a few hours of quiet. He may sometimes complain of pain, but it is usually referred to the knee. But, even in this early stage, if the child be stripped and examined carefully, the disease may be detected.

By stripping the child naked and standing him before you, either on the floor or table, and with his back towards you, the first thing noticed will be that the child bears his entire weight upon one of his limbs, the other being slightly bent at the knee and hip, with its natis lower and more flattened than on the opposite side, and the gluteo-femoral crease is less distinct and nearly obliterated at its outer angle, and is lower than the other. If you now let the child walk around the room, he may not limp sufficiently to attract your attention in this early stage of the disease. But when you bring him back to the position as first described, and let him stand a moment or two, you will find that he invariably resumes the position of sustaining his whole weight upon the sound limb.

You now lay him on his back upon a table, floor, or some solid plane, covered with only a blanket, in such a manner that his entire spine will be brought upon the plane, while a line is drawn from the centre of the sternum over the umbilicus to the centre of the pubis is crossed at right angles by a line drawn from one anterior superior spinous process of the ilium to the other. This can be done by placing your arm under the [illegible] ing the thighs until the spinous processes of the [illegible] the solid plane upon which the [illegible] the two lines ab[illegible] column is slightly [illegible] at right ang[illegible] hip-joi[illegible] th[illegible] suspected limb in your hand in such manner as to keep the spinous processes on the table while the other lines are at a right angle, you will observe that the well limb can be pressed down to the table so that the popliteal space will touch. The diseased limb can be pressed down nearly to this position ; but before the popliteal space touches the plane, you will notice that the pelvis becomes tilted, making a curve in the lumbar vertebræ, so that the hand can be passed between the child's back and the table.

In flexing the limbs, the well one can be completely flexed, so that the knee will touch the thorax. The diseased one cannot be flexed to this extent ; and before the knee will touch the thorax, the pelvis becomes lifted. Abduction and external rotation may, possibly, at this period, be carried nearly to their normal extent, without much pain. Adduction and internal rotation are much more limited. Compression of the head of the femur into the acetabulum by concussion at the knee, or pressure over the trochanter major, will give pain, providing the pressure is made so that the entire head of the femur shall sweep around all parts of the acetabulum, the pain being made manifest the moment the parts come in contact where the disease exists. Extension—very slight extension—in the proper direction gives instant relief. On measuring the thighs, even in this early stage of the disease, atrophy of a $\frac{1}{16}$, $\frac{1}{8}$, or $\frac{1}{4}$ of an inch will be found to have occurred.

If the disease be detected in this early stage and properly treated, I am satisfied, from extensive experience, that the great majority of cases will entirely recover, with perfect motion and without deform- [illegible] This fact alone, gentlemen, which I know to be a fact from [illegible] observation, is, of itself, a sufficient contradiction to the [illegible] ly expressed, that the disease was of constitutional ori- [illegible] ended in death, or if in recovery, with more or less [illegible] fect motion, or anchylosis. If the disease be [illegible] stage and properly treated, it progresses until [illegible] within the joint, and, in order to accommodate this increasing effusion, the limb becomes more flexed, abducted, and everted, or rotated outward, to unfold, so to speak, the capsular ligament, thereby enabling it to accommodate itself to the increased amount of fluid within it.

This is the second stage of the disease. The adductor muscles become very rigid and contracted under the influence of reflex irritation ; constant efforts are made to draw the thigh inward, which are unavailing, as it is impossible for the limb to yield to their tractile force, owing to the effusion within the capsule compelling it to assume the position above described.

This stage of the disease is attended by the most acute and agonizing pain, the slightest attempt at motion, concussion, or compression causing the most distressing torture ; even the jarring of the bed, stamping upon the floor, the slamming of a door, or anything that causes the least movement of the bed upon which the little sufferer is lying, may be followed by an increase of pain. At this period of the disease the attendants are frequently awakened at night with a sharp, shrill, agonizing shriek. The mother runs to the child—and probably finds it asleep ; she will have scarcely reached her .room before the same thing again occurs, and this will frequently happen during a single night. The inflammation of the joint produces reflex contractions ; the muscles are all on guard to prevent the joint from having the slightest motion. This incessant, constant, unremitting contraction of the muscles preys upon the child, producing exhaustion, until finally, through sheer fatigue, he may drop off into a moment's slumber, when, the muscles being relaxed, the limb falls down, motion is made in the joint, which causes such instantaneous pain that the muscles at once give a spasmodic contraction followed by the piercing scream to which I have just alluded.

Of course, at this period of the disease it can hardly be mistaken ; but upon stripping the child, and examining as I have directed that he should be examined in the first stage, he will be found to present, both in the erect posture and prostrate position, precisely the same appearances as in the first stage, only in a more marked degree, the chief differences being that the limb will be more flexed, abducted, and *everted*, and the joint more fixed; in fact, any attempt to move the limb in this stage of the disease is futile, the entire pelvis rolling upon the opposite acetabulum as if the diseased hip were anchylosed; and yet this anchylosis is but apparent, being wholly due to muscular rigidity.

If the disease be not arrested at this stage, it goes on to ulceration of the capsule, and effusion of its contents into the cellular tissue about the thigh, or else perforation of the acetabulum, and escape of the fluid into the pelvic cavity, pressing off the internal periosteum before it.

This is the third stage of the disease; and immediately the capsule is ruptured, and the contents escape, the limb assumes an entirely different position. It becomes *adducted*, *inverted*, straight at the knee, the pelvis on the diseased side becomes raised (whereas before, in the other two stages, it was lower), and the limb is shorter. The gluteo-femoral crease is higher than upon the opposite side.

If the rupture through the capsule is very large, so that the escape of its contents occurs rapidly, this change of form from that of the second to that of the third stage may take place in a single night. If the opening be small and fissure-like, and the contents ooze out slowly, it will require a longer period for the deformity to assume the position which it does in the third stage. In some cases, even when the capsule has ruptured or the acetabulum has been perforated, the limb will remain in the position of the second stage, owing to adhesions which may have occurred, or to the head of the bone being locked in the opening through the acetabulum. The pain, at this rupture of the joint, is very greatly relieved. It has been thought by many authors that at this period the limb was absolutely luxated from the acetabulum. The sudden change in the distortion from flexion, abduction, and eversion, with elongation, to shortening, inversion, and adduction, has caused this belief; but of the fifty-two cases I have exsected I have never seen luxation upon the dorsum of the ilium in hip disease except in one instance. The absorption of the head and neck of the femur, produced by constant pressure, diminishes its size; the acetabulum, being also pressed upon, has been eroded and absorbed. But while this interstitial absorption has been going on within the acetabulum, periostitis has been going on upon its upper and outer borders, throwing out new osteophytes, and the capsular ligament has been creeping up, increasing immensely the size of the joint, but still retaining within its embrace what is left of the head and neck of the femur. It therefore might be called a displacement of the acetabulum; but so long as the capsule surrounds the head and neck, it ought not to be called a dislocation. (As long as the head is within its capsule it cannot be called luxated.)

The pus or other fluid having now escaped from the capsule, the patient is greatly relieved, but the disease still progresses. Constant muscular contraction promotes interstitial absorption of the head and neck and the acetabulum, and the pus which has escaped into the cellular tissue burrows in different directions, forming abscesses, which open at various points, according to the position in which the child has been placed, and finally makes its way to the outer world, sometimes opening behind the trochanter, sometimes a long distance down the thigh upon the outer side, frequently upon its inner side; and even, as I have seen, if the acetabulum has been perforated, making its way between the internal periosteum, and forming an opening above the pubis; so that the third stage may be accompanied with external fistulous openings, and very great exhaustion from extensive suppuration.

Gentlemen. we now have here three cases typical of the three different stages of the disease I have endeavored to describe to you. We will first observe them walk. You will notice this little child, six years of age, whose trouble dates from a fall down six steps some eight months ago. The fall was followed by a slight limp some few days afterwards, which has continued at intervals from that time to the present. He has complained of but little pain, and that always has been in his knee. He has been to numerous institutions, and has had various opinions expressed in regard to his case ; some terming it rheumatism, others growing pains, and again others disease of the knee-joint, for which he has had iodine painted upon it as you see ; and it was not until a few days ago that he was suspected to have disease of the hip.* You see he walks around the room with scarcely a perceptible limp ; he stoops to pick things from the floor, bending his hip tolerably well.

The second boy which we here show you, ten years of age, fell from a horseblock four months since, striking upon his trochanter major, and the injury was followed almost immediately by a slight lameness. A few days' rest, and he seemed to be so much better that no further attention was paid to it. After some weeks he began to complain of his knee, became slightly more and more stiff in his hip, but did not complain sufficiently of his hip to attract attention to it, until he was brought to me two days ago in the condition you now see him. You observe that he cannot walk at all, but glides around the room upon one foot, first upon his heel, then upon his toe. You see how carefully he preserves his right limb from the slightest degree of motion. You will notice, when he stands, the limb is bent at the knee and hip much more than in the other case, and is apparently longer, and is extended in front of its fellow, and strongly abducted and everted. When he attempts to stoop to pick up anything from the floor, you will observe that he does not bend that leg at all, but sticks it straight out behind him, to prevent motion at the hip-joint.

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* A few days before Dr. Sayre saw this case, he was brought to the Clinic for Diseases of the Nervous System, at the College of Physicians and Surgeons, to have a " neuralgia " of the outer part of the thigh investigated, when I made the diagnosis of morbus coxarius. [E. C. SEGUIN.]

We now observe this other case, a little girl on crutches. She is about five years old, and fell off the stoop nearly a year and a half ago, but did not complain for some weeks after the fall, when the disease slowly developed, and has gone through its various stages, until it has reached its present condition. She is brought here to-day for the first time, and you see her condition. She is unable to walk except upon crutches, unless she glides along first upon the heel, then upon the toe, as in the second case. Her leg is shortened, and the foot, you observe, hangs over the instep of the opposite side. There has been no mechanical treatment of this case to prevent the deformity which you now observe.

We will now strip these children, and stand them together upon the table before you, that you may see the characteristic deformities of the disease in its different stages. We now turn the table, so their backs will all be towards you. It is a singular coincidence, gentlemen, that in each of these cases the disease is on the right side. This little fellow in the first stage, you will notice, stands firmly upon his left limb; and his right one, as you see, is very slightly flexed at the knee and pelvis, so as to receive no weight. The gluteo-femoral crease is lower than upon the opposite side, and his natis slightly flattened. His limb is very slightly abducted; but you observe that his feet are parallel—foot not everted. Now, look at the second little fellow; you notice how much more his knee is bent, as well as his hip, and his limb stands forward from the other, apparently too long; and you see his limb is much more abducted, and his foot *everted*, a positive evidence of effusion within the joint. His natis is more flattened, and the gluteo-femoral crease entirely obliterated. We will now pass to the third case. You see she requires her crutches to sustain herself. But her limb hangs against, and even over, the opposite one. When she stands erect, the pelvis on that side is elevated, and the natis rounded out. The gluteo-femoral crease is higher upon that than upon her well limb, and the leg is some two or three inches shorter than the other.

We now lay these children upon their backs, and commence with the first. You will observe as he lies upon the table, placing my hand under his knees, I get the line from the sternum to the pubis at a right angle to a line from one ilium to the other, and while the spinous processes still lie in contact with the table, I drop his left limb and make the popliteal space touch the table without any change in his spinous processes, or in the relation of the lines I have described. When I drop his right leg you will observe I go nearly to the table before altering those relations; but when I press his popliteal space down *to* the table his pelvis moves, and my hand can pass between his back and the table. I flex his left leg until the knee touches the thorax. The right one flexes to more than a right angle, when, you observe, continuing the flexion, his pelvis becomes lifted. Pressing his limb directly upwards in this instance seems to give him no pain. When I abduct and rotate his limb outward, and then press the limb upward, I produce pain, showing the necessity of careful exploration of the entire acetabulum.

We will now place a mattress upon the table and make the same examinations. You observe I bring his legs parallel with each other, perfectly extended, to all appearances, making the popliteal spaces touch the bed without, apparently, tilting the pelvis, the deformity being so slight that the spring of the mattress obscures it; and an examination upon a soft substance like this would, therefore, deceive you, particularly as in this case pressure upon the leg at the knee directly upward gives him no pain, the disease in this instance being in the inner part of the acetabulum. You can see how easily you could be deceived in this particular case, and that, probably, is the reason why he has passed through the hands of so many skillful men in the city without the disease being detected.

We now take the second child and lay him on the table, getting our lines as before described; and you observe, to obtain them, we are compelled to abduct, evert, and strongly flex his thigh. The least attempt at motion, in his case, is followed by intense pain and spasm of all the muscles, his joint, apparently, being as completely fixed by contraction as if there were genuine bony anchylosis; any attempt at motion, as you observe, tilts his pelvis so much that even on the mattress it can be noticed and his disease diagnosticated.

We now place this third case upon the table, and, to obtain my lines as before described, I am compelled to hold the diseased limb, not so flexed as in the second child's, but strongly adducted beyond the median line; in fact, as you perceive, a plummet dropped from the knee falls outside the lower extremity of the femur upon the opposite side. I can bring that limb parallel with the other, but the instant I do it her pelvis becomes tilted and the deformity takes place in the sacro-lumbar region. Many persons deceive themselves in this stage of the disease, thinking they have performed miraculous cures, correcting the deformity by mechanical appliances, when they simply have made the limbs parallel at the expense of a compensating curve in the lumbar region, no movement whatever having taken place within the joint. I caution you, gentlemen, against deceiving yourselves in this manner. Remembering always the lines I have laid down for your guidance, which must be as I have described them, that the pelvis and trunk may be in their proper relations to each other, then the position the limb assumes, while retaining these lines in their true relation to each other, gives the exact deformity, and indicates the extent to which the disease has progressed. You will, then, and only then, have the real deformity which exists at the hip-joint. If these lines are not retained in the position which I have described, any change which takes place in the position of the limb will be followed by numerous curves in different parts of the body as compensating deformities.

We now come to the treatment of this disease, which, of course, varies according to the stage in which we find it, and the reason why I have dwelt so particularly upon the diagnosis of the disease in its earlier stages is because, as I have before stated, by proper treatment at this time, in the great majority of cases, it will be followed by good results.

In the *first stage* of the disease, if the symptoms of inflammation be very acute, pain and tenderness very great, rest, absolute and perfect rest of the joint, is most essentially requisite. In the hearty, robust patient in vigorous health, leeches, cups, or other local depletion may be necessary ; ice-bags surrounding the joint often afford the greatest possible relief, while in other instances hot fomentation, the exact opposite, will give the greatest ease. There is no rule with which I am acquainted that will guide you in the application of heat and cold excepting by practically testing, and the one which you find affords the greatest relief, and is the most agreeable to the patient, is the one to employ. In addition to these local applications, extension should be made by means of a weight and pulley secured to the limb by adhesive strips and a roller. The adhesive strips should always extend above the knee to avoid traction upon this articulation. The pulley should be attached in some manner to the bed, the foot of which is elevated ten or twelve inches, to make the body a counter-extending force. If the bowels be constipated, cathartics, as a matter of course, are indicated.

This plan is to be pursued until the more acute symptoms have subsided ; but as it is a disease chronic in its nature, long confinement in a bed is injurious to the general health, and we must, therefore, contrive some mechanical appliance which will give extension and counter-extension, at the same time admitting motion of the joint while it permits the patient to take exercise in the open air.

In some cases, where the disease is very acute and the children very small, this is best effected by placing them in a wire cuirass; a modification of Bonnet's *grand apparèil* will be found very useful. When this instrument is employed, it is necessary that the child should be taken from it very frequently, and have all the joints carefully moved, otherwise too long continued rest of the joints may end in anchylosis. In moving the diseased joint, care must also be taken to hold the pelvis, and to make slight extension upon the diseased limb when motion is given to the joint. Perfect rest, long continued, even of the diseased joint, is decidedly injurious, as there is danger of it resulting in anchylosis; hence the objection to plaster-paris or any other fixed apparatus in the treatment of this disease. The disease is essentially within the joint, the capsular ligament not being involved; hence, all that is requisite is extension and counter-extension to prevent the diseased surfaces from being in contact, while at the same time motion is given to keep the parts uninvolved ir a healthy condition.

If the child is large enough to run around, with the thigh sufficiently long to give attachment to adhesive plasters, the short splint which I have used for so many years is altogether the best to employ, as it admits of flexion of the knee, and is, therefore, more comfortable to the child in the sitting posture. If they are ten or twelve years of age, and are too heavy to bear the weight of the body upon the instrument without breaking it, or there is too much tension upon the skin by the adhesive plasters, crutches will be necessary when the short instrument is used. If, however, the child's thigh is too short, and he is too small to receive a sufficient amount of extension by the use of the short splint, then the long splint, which I here show you, is much preferable, and, with it applied, the patient is able to take exercise without the use of crutches.

Before applying the short splint, which we shall use in this first case, it may be as well to describe it. It consists, as you see, of a curved cross-bar, surmounting the crest of the ilium, well padded on its inner surface, and to its two extremities is fastened a perineal band for counter-extension ; on its outer surface a ball-and-socket joint, from which runs an iron rod or bar down the outer side of the thigh to within about two inches of the lower end of the femur. This outer bar is divided into two sections, one running within the other, and gauged or controlled by a rachet and key, which can make it longer or shorter. At the lower extremity of this outer bar is a projecting branch going around to the inner surface of the thigh to receive the attachments of the plaster, hereafter to be described. Both of the lower extremities terminate, as you observe, in a cylindrical roller, over which the tags of the plasters are attached to the two buckles placed at the lower ends of the instrument.

In applying the instrument, it is first necessary to have the adhesive straps to which it is to be fastened properly secured, and this is done as follows. When using the short splint, night-extension is necessary, which is effected by means of weight and pulley : for this purpose a strip of adhesive plaster, to the lower end of which a stout piece of webbing is sewed, is placed on either side the leg, extending from the malleoli to *above* the knee, in order to avoid traction on the lateral ligaments of the knee-joint ; this is secured by a well-adjusted roller, leaving the pieces of webbing projecting for the attachment of the extending force. Next, for the application of the instrument, a triangular piece of plaster, in which are cut several slits converging toward the apex of the piece, is placed on both the outer and inner side of the thigh, first measuring with the instrument so that the tags which have been sewed to the apices of the plasters will exactly conform to the places of attachment upon its lower extremities. Having secured these with a roller, using care at the upper part of the thigh to reverse each alternate strip of the plasters in carrying round the roller, and with another turn taking in the other strips—braiding them in, basket-shaped—run the roller down the thigh again and sew.

In applying the instrument, first buckle on the straps at the lower extremity of the femur. Then pass the strap under the femur to the buckle at the outer side of the instrument for the purpose of keeping it in place. We now pass the perineal band around the perineum, and buckle it snugly, but not too tight. We next insert the key into the rachet and make gradual extension, until the patient is made perfectly easy, and until compression can be made upon the femur against the acetabulum without pain.

If the disease has arrived at the *second stage* before we see it, and the effusion is very great, which will be indicated by the abduction, flexion, and eversion of the limb—sometimes even fluctuation can be detected—the patient must be kept in bed and the extension applied to the limb exactly in the line of the distortion, which will be in the line of flexion and abduction. This line of extension is to be changed day by day, by slight degrees, until the limb is brought as nearly as possible to the straight position. Blisters applied occasionally over the joint may hasten the absorption of this effusion. Firm strapping with adhesive plaster around the joint and compression with a sponge and roller may also be applied for the purpose of aiding absorption ; of course, extension being used before this compression is employed. It is barely possible that the effusion may be so great as to paralyze the absorbents, and no treatment will decrease the effusion. Under such circumstances aspiration of the joint is not only advisable, but the proper treatment, and will be immediately followed by a restoration of the joint to its natural position, as you all saw in the case operated upon before you last Wednesday.

When the limb has been brought to nearly its normal position, then the treatment by the short or long splint, according to circumstances, is the same as in the first stage of the disease, the plasters being re-adjusted as often as necessary. Good adhesive plaster (Maw's, of London, I have found to be the best), properly applied, will frequently remain in position from two to four months, seldom requiring removal oftener than once in six weeks to two months.

If the disease has gone to the *third stage*, capsule ruptured, abscesses formed and not yet opened, it is necessary to puncture these abscesses at various points where they are nearest the surface to prevent the pus from burrowing. The limb then being adducted, the extension, as a matter of course, must be exactly in the opposite direction from what it would be in the second stage of the disease, and the limb gradually abducted until it is brought parallel with the other, when the splint, either long or short, is requisite, to be modified by the *abducting screw*, which I have been in the habit of using for many years with great advantage. In numerous instances, even when the disease has progressed to this stage, by the use of the splint the patient is enabled to improve the general health by out-door exercise, which frequently results in perfect recovery, and in some cases with a moderate degree of motion. The majority of the cases, however, that have arrived at this point before proper treatment has been adopted, are apt to recover with more or less complete anchylosis; in fact, anchylosis should be considered in this stage of the disease a very favorable termination.

In this third case we will apply the long splint, which differs from the short one described above in the following particulars :—

In the first place, it extends the entire length of the limb, receiving the weight of the body at a cross-bar under the foot, and two perineal straps and an iron girdle very nearly encircle the pelvis. In this case, where the adduction is so great and the joint so fixed, it will be necessary also to apply the abducting screw, and in some cases, where the inversion is very great, a screw for the rotation of the foot outward is also necessary, as in the instrument

I now show you. In this particular case we will apply the splint with the abducting screw added. The long bar, reaching from the pelvis to the bottom of the foot, is hollow, and has another bar running inside of it furnished with a rachet and key, by which we make extension, and which is locked in the same way as the short splint. The cross-bar at the bottom of the instrument (similar to Taylor's) is covered with leather to keep from making a noise on the pavement while walking, and a strong leather strap is passed beneath two iron rods above this latter for the purpose of buckling on the adhesive strap upon the leg to make extension.

In applying it you take two strips of strong moleskin adhesive plaster from two to four inches in width, according to the size of the patient, and the entire length of the limb, the upper extremity of the plaster being divided into strips for two or three inches. Strong webbing, an inch or two in length, with buckles, is sewed fast to the lower extremities of the plasters. These plasters are then placed on either side of the leg in such manner as to leave the buckles a little above the ankle-joint, and secured by a snugly-adjusted roller, so applied as to leave the tags with buckles attached hanging loose, the roller being carried up over the knee, and as far up the thigh as can be done with convenience, when the upper split ends of the plasters are reversed and braided in with the roller as it turns down the thigh, securing it smoothly. The stocking is then pulled up on the foot, holes having been cut on either side for the buckles to pass through, and the shoe applied with holes cut through it in the same way.

The instrument is now placed on the outer side of the leg, and the cross-bar at the bottom brought in front of the heel of the shoe, and securely buckled to the tags above described. The pelvis-belt is next brought around the hips, and secured by the buckle upon the opposite side, and the perineal bands attached as firmly as need be. The knee-pad band is then slipped up or down until it is made to rest opposite the knee, where it is passed round the leg and buckled fast. Extension is now made by the key upon the rachet until free compression is borne without pain. The abducting screw is then to be used, and daily increased, for the purpose of abducting the limb.

If the limb be strongly flexed, an additional power is applied at the posterior part of the instrument at the knee, running up the back of the thigh, and secured to the posterior portion of the pelvis-belt, and made tighter as occasion may require for the purpose of extending the limb. This latter strap should be elastic, for the purpose of keeping up a constant tractile force, and at the same time allow of flexion when the patient wishes to sit down. A fixed or leather strap, as used by Taylor, prevents any motion whatever at the hip, and simply anchyloses the joint.

By this means many cases that have gone to the third stage of the disease may in the course of time recover without exsection, as you have seen, with tolerably good form and a moderate degree of motion, without any further operative procedure.

If, however, notwithstanding your treatment, the disease progresses, suppuration increases, the joint becoming more and more impaired, showing a case of progressive caries, we then have no remedy except in *exsection*.

Nature's only way of curing these cases after they have arrived at this point is by the slow exfoliation of the carious bone, and, if this is limited in amount, she is often successful; but if involving the entire head and neck of the femur, with more or less of the acetabulum, as it frequently does, the process is a very tedious one, and the patients often succumb before nature completes the cure; and in the most favorable cases healed by kind nature in this way, they have been left with permanent deformity, and with a very much less useful limb than those which have been cured by exsection. I have now performed this operation over fifty times, and can, therefore, speak with positive assurance upon the subject.

This operation is very simple, indeed, and attended with no danger whatever. The patient being anæsthetized and laid upon the well side, an incision is made from a point midway between the crest of the ilium and the top of the trochanter major, the knife carried firmly down to the ilium, and drawn with a single sweep downward and outward over the posterior edge of the trochanter major, and then curved forward and inward, making a crescent-shaped incision of some four to six inches in length, according to circumstances, and carried fairly down to the bone its entire extent. The wound is then held open with spatulas, and a narrow firm-bladed bistoury is carried around the femur just above the trochanter minor at right angles to the first incision, and divides the periosteum in both directions as far around the bone as can be reached, one-half or three-quarters its circumference. By this circular division of the periosteum you avoid the danger of tearing it off from the femur below the point where section is to be made. If the first incision has not divided the periosteum completely, then carry your knife again through the first incision from the top of the trochanter major down to this cross-incision just described, pressing it firmly through the periosteum down to the bone. The periosteal elevator is then placed in these two triangles, and the periosteum peeled off from the trochanter, carrying with it necessarily the muscular attachments to it. This can be very successfully done until you reach the digital fossa at the neck of the bone and behind the trochanter, where the blade of the knife will be necessary to divide the tendinous insertions of the rotator muscles. The capsule being freely opened, the head of the bone will now be easily thrown from the acetabulum, by strongly adducting the limb and depressing it, thereby tearing off the internal portion of the bone from its lining periosteum, when the finger can be glided around the bone, and with a finger-saw it may readily be removed below the trochanter major. By this means the periosteum will not be peeled off from the bone below the point of section with the saw, as is too often done by luxating the bone too forcibly.

If upon the first section it is found that the caries has extended still farther down the femur, you can very easily separate it from its periosteal attachments, and whatever amount of bone is necessary can be removed in the same manner with the saw. Under no circumstances should bone forceps be used in the section of so large a bone. The trochanter major should always be removed, even if it is not diseased, as otherwise it would occlude the opening, and prevent the escape of the discharge; and by peeling it from its periosteum, as I have before described, the attachments of the muscles are all left for future use.

When the head and neck have thus been removed, you have a fair opportunity for exploring the acetabulum, and to remove all the carious or necrosed bone by scraping and gouging. If the acetabulum be perforated, which I have frequently found to be the case, with a little care the necrosed bone can be broken off down to the point where the periosteum is attached. I have only in one instance found the internal periosteum perforated.

After washing the wound carefully with warm water, fill it with peruvian balsam; a round small plug of oakum, long enough to reach the very bottom of the acetabulum, is inserted, and left dependent from the wound. The upper and lower ends of the incision are then brought together by stitches and adhesive plaster, and the patient placed in the wire cuirass which has been constructed for this purpose, with a window opposite the place of incision. As it is of the greatest importance that this dressing should be done with care, I will describe to you my mode of doing it.

The cuirass being properly prepared and well padded, the patient is laid in it so that the anus is opposite the opening and free from any possibility of obstruction, when the well leg is the first to be dressed. By making it perfectly straight and screwing up the foot-rest until it is brought firmly against the heel of the patient, having a pad between the foot and the rest to absorb the perspiration, the instep is then well padded with cotton or a blanket, and a roller is carried firmly round it and the foot-rest, running up over the limb; but before going over the knee a piece of pasteboard, or leather, or several pieces of folded paper, are placed over the leg, knee, and thigh, and the roller carried firmly over this extemporized splint for the purpose of preventing the slightest bending of the knee, when the roller is carried up the entire length of the thigh, around the perineum and over the outer arm of the instrument, and several times back through the perineum, and then across the pelvis, by which means the well limb is made a firm counter-extending force.

Two strips of adhesive plaster from two to four inches in width, according to the size of the patient, are then placed upon either side of the operated limb and secured with a nicely adjusted roller over the foot and up the limb and thigh as far as the abscesses on it or the wounds will permit, being careful to leave a sufficient length of the plasters, at the lower extremity, free for the purpose of applying them to the foot-rest when extension is made, and firmly secured by a well-adjusted roller. The foot-rest is then screwed up to meet the heel of the shortened limb, and these strips of adhesive plaster are brought down around the foot-rest and securely fastened. The foot-rest is then extended by the screw slowly, and gradually, at times waiting a few moments for the muscles to yield, which have been so long contracted, until the limb is brought down to its full extent. It sometimes happens that, from long contraction of the adductors and the tensor vaginæ femoris, subcutaneous section of those tendons and fascia will be requisite before the limb can be brought to its proper position, even after the head of the femur has been removed. After the limb is brought into this position a roller is carried from the foot over its entire surface; a large wad of oakum is placed around the wound to absorb the discharge, and the roller is carried firmly over the wound, inner surface of the thigh, and around the pelvis. I place great importance upon this latter part of the dressing, as we thereby compress the tissues, and prevent the burrowing of pus, the oakum, which has already been placed in the wound, allowing of free drainage, no matter how tight the roller may have been applied.

Immediately after the patient is dressed in this way, and has recovered from the anæsthetic, he is capable of being stood up against the wall, or riding out in a carriage or boat, and can take his daily exercise in this way. I have, in several instances, had them removed a long distance, some miles, in fact, within an hour of the operation and without the slightest inconvenience or pain. This dressing will probably not require to be changed for from 48 to 60 hours, or until sufficient secretion has been formed to moisten the dressings, when the oakum plug can be removed without hemorrhage. If this dressing does not come away easily, warm water injections will readily float it out. The wound, made clean, is again filled with peruvian balsam and dressed as before. After this it may require dressing once or twice a day, according to the amount of discharge, and the child should be removed from the entire instrument as often as is requisite. The well leg should be removed from the instrument at least once a week, and free movements given to all the joints, ankle, knee, and hip, otherwise we may anchylose them, although they are not diseased. The wire cuirass should be used from a month to two months, according to necessity, after which the patient can be put upon the long splint and allowed to exercise, thereby increasing his prospects of perfect motion of the new joint.

In many of the cases which I have exsected, the motion has been as perfect and complete as in the normal joint, and in one case, Adolph Rosell, the motion is greater in that joint than upon the opposite side, and the limb less than a quarter of an inch shorter than the other, although it was sawed three inches below the top of the trochanter major; the head and neck having been entirely absorbed, and the acetabulum perforated. Only one of my exsections has recovered by anchylosis, and that was from neglect in the after-treatment, I never having seen the patient after the operation for two years, and the gentleman who had it in charge having no experience in the treatment of this class of cases. All the other cases that recovered have more or less good motion, and infinitely less deformity than those which have recovered by nature's process.

If the surgeon has not the convenience of obtaining the wire cuirass, the operation can be made just as successful by applying extension and counter-extension, while the patient is in bed. Of course, they lose the advantage of out-door exercise and fresh air, which in many instances will be found to be of vital importance.

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ACUTE RHEUMATISM IN INFANCY AND CHILDHOOD.*

BY

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Gentlemen:—This little girl, three years old, was presented to you last Saturday, the day after her admission. She came with a history of a pharyngitis, for which she had been treated with so much success, that the mucous membrane of the fauces exhibited but few remnants of that disorder. Still, you remember, the child appeared to suffer. There was dyspnœa, respiration about forty in a minute, temperature 104°, pulse 160 and more, respiration over the upper lobe of left lung diminished, in some places harsh, approaching the bronchial, some little bronchophony, and corresponding dullness on percussion. At the same time, we were told that there was slight œdema of the feet. From these symptoms pueumonia in the left upper lobe was diagnosed. One peculiarity, however, required further study, viz., that although the number of respirations and of pulsations were both increased, the proportion between the two was normal; whereas, with pueumonia, there should be an excessive number of respirations. We looked farther for an explanation of the undue frequency of the pulse. It was found, as we thought, in the previous history of the child. When nine months old, she had hooping-cough, which lasted a long time, was complicated with fever, and succeeded by frequent and protracted attacks of coughing. Thus we explained the frequency of the pulse by the presence of a chronic pulmonary infiltration, in consequence of which the heart had to overcome the obstacles in the pulmonary circulation by accelerated contractions. Besides, the long-continued venous obstruction, and the general hydræmia of the patient appeared to explain the œdematous swelling of the feet. The treatment was to consist of a daily dose, or two, according to circumstances, of five grains of quinia, with a sufficient number of doses of digitalis to reduce the pulse; for we feared that the heart might be exhausted by its over-exertion. There was still another reason for this medication. In the hasty examination we were permitted to make, the child being very sick and the amphitheatre cold, I noticed that the mitral systolic sound was rather prolonged and muffled. Still, no diagnosis was based upon that fact at the time, although that of endocarditis suggested itself. I ordered the digitalis to enable us to make a diagnosis of the cardiac disorder, if any there were; for a murmur will frequently be heard when the heart's action is rendered slower. The blood-wave being smaller, when the heart moves fast, irregularities of the valves may not be detected, while as soon as a larger amount of blood passes over the diseased surface, the morbid sounds become distinct. On the next day, the general condition of the child was much improved; temperature 101–102½, dyspnœa not so marked, but a distinct murmur was heard over the mitral valve, taking the place of the first sound. *Endocarditis*, with *incompetency of the mitral valve*, suspected on the previous day, was then diagnosticated in addition to the *pueumonia*. Greater attention was then paid to the œdema of the feet. We learned, as part of the child's history, that not only had the œdema existed before the dyspnœa set in, but also that the patient had been unable to walk for a number of days, and passive motion had been painful, both in the ankle and knee-joints. This important fact finally completed our diagnosis.

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* Delivered in the amphitheatre of Bellevue Hospital, February 13th and 20th, 1875.

We had to deal with acute articular rheumatism, rheumatic endocarditis, and pueumonia, occurring contemporaneously or in quick succession. To-day the child appears much more comfortable than last week. Temperature averages 101–102, reached 104 but once all the week. The symptoms of acute pueumonia have nearly disappeared, and no new attack has taken place, dyspnœa is moderate, but the bellows murmur is very loud, both anteriorly and posteriorly, both right and left. As there is no unusual amount of venous obstruction, the external veins not being much dilated, and no general œdema exists, we have reason to believe that this loud murmur results from friction of blood, not only over the mitral valve, but over the roughened surface of the entire endocardium, also involved in the morbid process.

Look at this other patient. She is a girl of ten years, well built and robust. She denies ever having had pulmonary disease, but reports that for six weeks past she has suffered from severe pains in her limbs, particularly in the knee and ankle joints; has been confined to her bed for a fortnight, and now cannot walk without pain; has had a slight, short cough for weeks, which has been harder and more protracted during the last week; finally, during the last five or six days, has raised blood: yesterday as much as a cupfull. Altogether, she feels very ill. Upon examination, we find considerable dullness over the upper lobe of the right lung, both anteriorly and posteriorly, with some bronchial respiration above, greatly diminished vesicular murmur, and some fine rales; large moist rales are disseminated over the left lung. Finally, and most noticeably, we discover at the heart roughness of the first sound at the pulmonary orifice, and prolonged to the left, in the course of the pulmonary artery. The diagnosis is clear; *acute articular rheumatism, endocarditis at the orifice of the pulmonary artery, solidification of the right lung, and pulmonary hemorrhage.* Now an affection of the pulmonary artery is extremely rare in rheumatic endocarditis, particularly when no other locality of the heart is affected.

We ought, therefore, to inquire whether, in our case, this stenosis be not perhaps congenital. The age of the patient renders this question more pertinent than would be the case were she more advanced in years. For in congenital disease of the heart, it is precisely the pulmonary artery which is most often affected, and the lesion may persist later in life,—causing symptoms of heart disease with or without cyanosis. In order to decide the question, whether the existing lesion be congenital or acquired, I offer the following considerations :

In congenital stenosis of the pulmonary artery, either the foramen ovale or the ventricular septum must be found patent. In neither case is considerable hypertrophy of the heart a necessary consequence. If, however, a considerable hypertrophy of the right heart is found, with the usual change of the position of the heart in such cases, you may conclude that the anomaly is acquired, and thus it is here. The heart, as measured by percussion, is twice its normal size, and this hypertrophy is confined to the right ventricle ;—the percussion sound is dull to about a half inch beyond the right margin of the sternum, but not beyond the line of the nipple towards the left ; and from the pencil-marks made while I am percussing, you will perceive that the heart is in an oblique position. Our case then is one of the exceedingly rare ones of endocarditis, wholly or mostly confined to the right side, and to the pulmonary orifice exclusively.

But its interesting features are not yet exhausted. Is pulmonary hemorrhage such as our patient describes a frequent occurrence in a recent case of pneumonia ? Is it frequent in childhood ? Neither the one nor the other. What then does it mean here ? It means that some of the deposits in the pulmonary orifice have been torn off, once or repeatedly, that the mass was carried into the lungs, and that both the pneumonia and the hemorrhage are the results of an embolic process and infarctus. Thus, the second exceptional circumstance is in close dependence upon the first, and both render the case one of rare interest.

Third case: This little girl is four years old. She has been well until four weeks ago. At that time she refused to walk or stand. The ankle-joints were swollen and painful to the touch, and the knee-joints and wrists were in the same condition. The history is not very clear, as we have no other report but that of the mother, and that only contains the fact that the ankle-joints were first affected, the wrists next in order. Both are still swelled and painful. A rather loud bellows murmur replaces the first sound of the heart over the mitral region. These details suffice already for the diagnosis, *Rheumatic Polyarthritis* * *and Endocarditis*. The patient has a short, hacking cough, and is said to suffer from frequent attacks of nose-bleeding. In the lungs no abnormal respiratory sound is heard except some mucous rales. You know that slight pulmonary œdema and bronchial catarrh accompanied by this peculiar short cough, are frequent, indeed, almost inevitable consequences of mitral incompetency. To the same lesion is due the epistaxis, which never occurred before this sickness. It is the result of the retardation of venous circulation in the copious and loose connective tissues of the nares, than which there is none more richly supplied with blood-vessels. In this connection, take it for granted, as a general experience, that large numbers of cases of epistaxis in infancy and childhood are the results of mitral insufficiency, mostly attended by general hydræmia.

What are the elements common to all these cases? In all exist synovitis of a number of joints, and endocarditis, while other symptoms or complications vary. The membranous connective tissue is inflamed and secreting over many and large surfaces. The peculiarity common to both the synovial and the serous membranes is the large number of blood-vessels, and the absence of glands. Their difference consists in the absence, on the interior of the synovial membrane, of the dense layer of epithelium which covers both the serous and mucous membranes. Therefore, the

* πολυς numerous, αρϑρον joint.

capillaries of the interior of the synovial membrane flow not below, but between the cells, a large number of which are mixed with the connective tissue. The intima secretes synovia, not from glands, not from transformed epithelium, not from blood-serum, but from the nutritive lymph pervading the connective tissue cells and interstitial spaces. Its secretion is easily induced; motion of the joint is sufficient to increase it. Local irritation is a ready cause for hyperæmia, hyperplasia, loss of superficial cells, and increased liquid secretion similar to that on the serous membrane. Such local irritation may be simply confined to one joint, as in traumatism. or multiple, and extending over a number of joints at the same time. In the first case we speak of a monarthritis,* in the last of polyarthritis. Polyarthritis—inflammation of many joints—cannot result from an injury, it must be attributed to some general and diseased condition, which, from its very generality, must be sought either in the vascular or nervous systems.

In regard to the first, it is remarkable how various are the specific blood diseases that may be accompanied by multiple joint-disease. Scarlatina, variola, pyæmia, and puerperal fever are the best known. In the first, an erythematous inflammation seems to be determined by an elimination of the poison upon the surface of the synovial, analogous to that taking place upon the skin and digestive mucous membrane. In variola, where endocarditis has been recently described, articular swellings are either due to hemorrhage (analogous to those in hæmophilia), or to an effusion of pus, and these are identical with the arthritis of traumatic or puerperal pyæmia. Arthritis is well known to be one of the most dangerous symptoms of pyæmia, and in the purulent effusion which it determines, is in striking contrast with the serous or serofibrinous effusion of a rheumatic polyarthritis. This effusion, which sometimes precedes pain, need not be inflammatory in the beginning; that is, there need not be in the beginning the charac-

*μονος single.

teristic process in the cell elements. Thus far it may be compared with the rapid effusion of pus in generalized purulent peritonitis, where the blood is overcharged with white blood-corpuscles, and the process is accompanied with paralysis of blood-vessels. It appears, therefore, that the presence of foreign matter in the blood constitutes a powerful predisposition to polyarthritis. This fact, derived from observation of diseases other than rheumatism, has been taken as a strong support to the theory, that in rheumatism some foreign material does circulate in the blood, be that lithic acid, lactic acid, or something else.

Still, the matter is not so easily settled. There is a great difference between pyæmia, scarlatina, and rheumatism, in their nature, and in their results on secreting surfaces. In pyæmia we know pus to be present, and pus is passed through the blood-vessels. In scarlatina we presume, almost know, that there is a foreign body in the blood, although Recklinghausen's bacteria theory is neither proven by him, nor confirmed by others. This foreign material is not visible as yet; at all events it cannot be compared to that in pyæmia in size or character. What do we see as the result of this material irritation in its synovial and serous eliminations? The rheumatic effusions of scarlatina are not always purulent ; on the contrary they are mostly serous ; somewhat thicker and darker in many instances, it is true, but purulent in but few instances. Still, pus is found sometimes in the synovitis and periostitis of scarlatina. But where is pus not found? Is it not the legitimate result of any obstruction of the circulation, as soon as leucocytes have a chance to escape? and are they not found in almost all and any effusions? Beside them, and pretty normal effusion of the surfaces, nothing is known as yet to exist in scarlatinous eliminations. Where then is its peculiar foreign material? If we assume it to be present in the effusion, let us not forget that such assumption cannot yet rank as a fact in the building up of a theory.

And now, of what nature is the effusion in rheumatism? Purulent? by no means. Additional leucocytes may be found in the serum of any effusion, as stated before; but the consistent character of the synovial and serous effusion in rheumatism is markedly watery and deprived of solid admixtures. It compares somewhat with copious effusions from large surfaces of mucous membranes. The mucous membrane of the intestinal tract will secrete a similar liquid by the gallon; that of the vagina even by the ounce or pound.

Thus, while the act of pouring out depends on the condition of the surface, which acts as a sieve, the nature of the morbid process underlying it cannot be explained by its result, particularly as long as the constituents of the discharge, though changed in proportion, are given off by the normal organism. The fact of purulent discharges taking place in pyæmia never solved the problem of its nature, and what we know of the nature of scarlatinous effusion never taught us the essence and origin of scarlatina; and it is equally certain, that the nature of rheumatic effusions never exhibited its causes. And as to changes in the blood in rheumatism? Neither the lactic acid, nor the lithic acid have ever been shown to exist; not even in the liquids effused by the effect of vesicatories have they ever been found.

It is much more rational to assume that some changes in the blood-vessels must coexist with the multiple fluxions which constitute the fundamental phenomena of the disease. I have already alluded to that form of arthritis—if it can be claimed as an arthritis—which occurs in hæmophilia, a disease in which the coats of the blood-vessels are congenitally altered or imperfect. It has been suggested that an alteration in the lining membrane of the vascular system was itself the original cause of the changes which almost certainly take place in the course of the disease. Thus, in rheumatism also, the cause of the various effusions would lie, no matter what its original source will be found to be, on the whole surface of the intima of the blood-vessels, from the endocardium to the smallest artery or vein, even the vasa vasorum. Endocarditis, then, would not be the complication of rheumatism, but its highest and most developed expression. I shall return to that question, and then you will see why it is that principally the left side of the heart, and principally the left vertricle, and principally again the atrio-ventricular orifice and mitral valve are affected.

The influence of the nervous system upon the condition of joint effusions is seen both in acute and chronic diseases. Hysterical arthralgia, however, described by Brodie, Stromeyer, Esmarch and others, does not fairly belong here. But recently Charcot discovered profound organic lesions of the articulations as a consequence of various diseases of the spinal cord. Besides, fatigue and exhaustion, prolonged lactation and an irritable nervous system, are just as many sources of predisposition to rheumatism. And the fact that warm bathing, tonics and nervines exert frequently a wholesome influence in rheumatism, appears to point in the same direction.

There is no proof, however, for the assumption that rheumatism is nothing but a neurosis. Dr. F. Lente (The Neurotic Origin of Disease, New York, 1875) says of J. K. Mitchell's tendency in that regard : "All of his cases are such of organic disease of the spine, or injury to the medulla, except one" (p. 9). This one case, however, claims only that it got well after bleeding, cupping over the spine repeated twice, and the administration of salts and magnesia ; nor does Dr. Lente's own case appear more conclusive, for the lady of eighty-three whose case he reports (p. 14), appears to have suffered from general thrombosis. She had senile gangrene, inflammation of both wrists, and cerebral symptoms.

If, then, the general nervous system be not called upon for a direct explanation of facts, what would you expect to take place when a sudden change of temperature affects the surface of the body? The irritated cutaneous nerves exert their reflex action upon the vaso-motor nerves, the superficial blood-vessels become contracted, their contents are suddenly driven below the surface into the vascular system of the large viscera, or of the serous and mucous membranes. Voluntary muscles and lungs are in constant expansion and contraction, and are, therefore, not very apt to be inundated. But the surfaces of mucous and serous membranes, offering less resistance, are the receptacles into which the blood chased from the surface of the skin is suddenly crowded together. The lymph contents are disturbed, epithelial cells softened and darkened, and secretion of lymph, and serum, and, in some cases, migration of white blood-corpuscles may take place. It depends on circumstances, previous disease, or vulnerability, whether the result will be a "catarrh" of a mucous membrane, or a "rheumatic" secretion of synovial membranes, just as it depends on individual disposition whether the mucous membrane of pharynx or intestine, or bronchi, suffer most after wetting of the feet, or similar occurrences.

This is not the only mode by which sudden contraction of the blood-vessels proves dangerous. The surface of the body, fourteen square feet in the adult, less absolutely, but more in proportion to height and weight, in the child, is the principal road through which the system gets rid of its heat. The cooling process is dependent upon a normal and sufficient cutaneous circulation. Sudden contraction of the blood-vessels means sudden accumulation of heat in the body; that is, "fever," with its consequences on the structure of tissue. As early as 1852, Virchow described, as the result of fever, parenchymatous inflammation with increase of nuclei, and indistinctness of cell contents. In 1864, Zenker described in the muscles of persons suffering from typhoid fever, a waxy degeneration, beginning in the connective tissue of the muscles, of simply degenerative, non-inflammatory character. His observations are correct, his explanation of the process is insufficient. For the term "degenerative" process does not necessarily involve a result from, or complication with, inflammation. Thus, traumatisms and trichinosis determine waxy degeneration and cell proliferation at the same time. In accordance with this, Waldeyer looked upon fever as a constant cause of both passive changes and cell proliferation, and Popoff has but lately (1874) described, as the results of increased temperatures, all the above changes. They are principally found in the diaphragm, recti abdominis, and arteries, in all feverish diseases, such as scarlatina, variola, measles, typhoid fever—and principally in those which exhibit the highest temperatures, such as scarlatina. They are probably a principal cause of chronic endocarditis, and may be also of chronic inflammation of almost any organ. At all events, we have good reason to believe that organs like the synovial and serous intimæ, immensely stocked with cells, and swimming in blood and lymph, are very apt to be affected by heat alone so produced, or rather accumulated, by the sudden contracting of the surface circulation.

A third cause of polyarthritis may not be general, but is frequent, viz., previously contracted endocarditis, with soft fibrinous masses deposited on any part of the endocardium. These, detached and swimming along in the circulation, may form emboli. In the muscles, they may remain undiscovered; in the brain, they may produce paralysis or chorea; in the joints, attacks of arthritis; in the lungs, infarctus and hemorrhage; and in the skin, ecchymoses in the shape of either purpura, or peliosis.

Thus you have a clue to many of the points of interest in acute rheumatism, both of adults and children. Children! Is rheumatism a frequent affection in childhood? Most books say No, the age of five or seven years is considered by many the earliest period of its occurrence, and a large number of them admit these only as rare exceptions in infancy. Two cases at a very early age, nine weeks and seven months, both by Staeger, have been reported in the journals. As a rule, however, you will find, that the frequent occurrence of rheumatism in infancy and childhood is resolutely denied, although every practitioner will have plenty of opportunity to come across non-congenital heart diseases at an early age. You have to-day seen three cases of the disease, two of which came here with a different diagnosis, and several have recently passed under your notice, while numerous old cases of endocarditis have presented themselves in the course of a short time. Thus, you are prepared to disbelieve the axiom that rheumatism is rare in infancy and childhood, and, on the contrary, suspect its rather frequent occurrence.*

While, however, it is frequent, it is rather different in many respects from the symptomatology of the same affection in the adult. I shall not occupy our time with a recapitulation of what you know from your lectures and other clinics. For this clinic has been established for the purpose of exhibiting the differences in the symptomatology, etiology, pathology, and therapeutics of the diseases of infancy and childhood from the same class of disorders at an advanced age, and so of forming a sort of comparative pathology and therapeutics. There are several peculiarities, which I shall enumerate briefly: In the rheumatism of children, the swelling of the joints is often but trifling, and sometimes disappears after a short time. The pain corresponds with the rapidity and quantity of effusion, and is, therefore, not always excessive; it is even trifling, and easily overlooked in rachitical individuals in whom flabbiness of synovial membranes and ligamentous apparatus are two permanent characteristics. Redness is but slight, or does not exist. Temperature is but rarely very high, as long as polyarthritis is the only symptom, and sometimes even low, after the first attack of an acute endocarditis has set in. Increase of temperature may often appear suddenly, and can sometimes be traced to an embolic process.* Unless there is endocarditis, respiration is in proportion to the fever; perspiration is not copious, urine not scanty, not often loaded with uric acid. On the contrary, it is frequently copious (particularly in early complication with mitral incompetency and venous obstruction), and pale. The course of the disease is perhaps still less regular than in the adult. It may last a few days, or many weeks or months. It may depend on hereditary peculiarities in the structure of the synovial intima or it may be induced by neglect of skin, diet, etc. The relation of the sexes to each other as met with in adult rheumatism does not hold good in the infant or child. Exposure being a frequent cause, men will suffer more frequently than women. Of children, however, I have, I believe, met with more female than male patients. Our patients to-day, are all of that sex, and the larger number of our heart diseases are also in girls. Visceral so-called "complications" are as frequent in the young as in the old, indeed much more so. Pneumonia and Bright's disease are rare; the tendency is rather in the direction of the mucous and serous membranes. Pharyngitis, laryngitis, bronchitis, peritonitis, are met with, pericarditis and pleuritis are not at all rare. But you will find, that as well in these "complications" as in the original synovitis the secretion is more serous than fibrinous. Therefore, friction sound is exceedingly rare in both rheumatic pleuritis and pericarditis of infancy and childhood.

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* In this connection I may, however, add that uncomplicated, muscular rheumatism is rare in young children. In most cases, where it appears to be present, the muscular pain can be easily explained. In torticollis, there is often an affection of the spinal column, or a hemorrhage in the sterno-cleido mastoid muscle. Roger compares the torticollis of infants with the lumbago of adults, which is often traumatic, or the result of rupture of muscular fibres. Gubler found articular rheumatism, endocarditis, and chorea some time after muscular rheumatism. "Growing pains" are probably of rheumatic origin, in not a few instances.

After this rapid review, let us look into some special facts. I said that the urine is not so red, not so scanty, as in adults. This corresponds somewhat with the physiological conditions of the renal secretion in the young. In a child of three years the proportion of the weight of the kidneys to that of the whole body is 1:146; in the adult, 1:230. Lecanu found the whole amount of urine, in a child of three or four years, in twenty-four hours, 225–325 grammes (gr. = 16 grains). Scherer, in one of three and a half, 755; Rummel, in a boy of three, 885–904; in a girl of five, 698–722.

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* The child presented at the head of the list, had, on the 14th, P. M., a sudden increase of temperature to 104¾. At my visit the 15th, we found the systolic murmurs less, the first sound setting in in a normal manner, but terminating in a murmur. Evidently a segment was torn off, and deposited somewhere as an embolus, as yet we do not know where.

A kilogramme (two pounds) of the

Adult's	**body**	**discharges,**	**29.5**	**grammes of Urine,**	**0.420**	**Urea,**	**1.101**	**Solids,**	**28.4**	**Water.**
Child's	**"**	**"**	**47.4**	**"**	**"** 0.810	**"**	**1.515**	**"**	**45.9**	**"**

in twenty-four hours. Salts are also increased in the child; mucus, extractive materials and uric acid are less by about one-half. The younger the child, the larger the proportion of urine and water, the smaller that of uric acid. According to Uhle the secretion of urine, urea and chloride of sodium, is treble that of the adult, uric acid being less in proportion. The urine of the newly born has a specific gravity of but 1005 or 1007. The amount of uric acid is in an inverse ratio to that of urea. Fever increases urea, according to Bartels, but not uric acid, as long as respiration is unimpeded. Therefore, copious deposits in the straight lobules of the newly born, so commonly met with up to an age of three weeks, are the result of insufficient supply of oxygen, and diminish rapidly where respiration is speedily and thoroughly established. They are found more in babies who have died of, or with asphyxia (Gerhardt, Dis. of Children, p. 4). The urine is particularly pale and light where mitral insufficiency is an early symptom of rheumatism, or where hydræmia is an early complication. This is to be feared because, as I have stated before, salts and nitrogen are rapidly eliminated through the kidneys, and besides, even in normal conditions, the amount of carbon eliminated through lungs and skin is almost twice as large in the child as in the adult (Scharling). Moreover, the fever, which would increase the amount of eliminated solids, is generally not excessive, and if high, seldom of long duration. The lesions required for effusion, and corresponding diminution of temperature, are generally not so thorough in the child as in the adult.

One more remark on the inflammation of joints in the child. Both traumatic and idiopathic inflammations are frequent. In infancy and early childhood there is less exposure, and therefore one great cause of rheumatism is eliminated. At that period of life the mucous membranes are more apt to suffer. The smaller joints are not so easily, or so frequently affected as the larger ones; thus, those of the maxilla, sternum and vertebræ are seldom the seat of inflammations. At any age the joints of the lower extremities are more liable to disease than any other, because their synovial membranes are larger, and because morbid materials circulating in the blood are more easily deposited in the extensive network of blood-vessels covering the intima. Thus it is that the pyæmia of the newly born is so very apt to cause suppurative arthritis of the knee-joint. Finally, the rapid growth of the synovial membranes, and of osseous tissue at the epiphyseal line of the bones, inside the joint, constitutes an important predisposition to pathological changes.

The cardiac manifestations of acute rheumatism exhibit in children a number of peculiarities. Their anatomical condition must necessarily be similar to that in adults. In both adults and children the mitral valve is most commonly affected; pericarditis comes next in frequency, lesions of the aorta next, and myocarditis last. That endocarditis of the right heart is very rare indeed, I have stated before. In all ages the origin of the valvular affection is the same. The valves are simply duplicatures of the endocardium covered with one or two layers of pavement epithelium, and joined to each other by elastic and connective tissue. The incipient stage of valvular inflammation consists in the production of a nucleated blastema with elongation and new formation of blood-vessels and hyperplasia of the connective tissue. Later, the epithelium exfoliates, and fibrine is deposited on the abraded surface. The valve may thus harden and retract, or else in certain cases soften, yield to blood pressure, and permit the formation of aneurysms, or even perforation. Similar changes may occur over the whole, or part, of the endocardium.

What is the relative frequency of heart disease in rheumatism in infancy and children? Most authors agree upon one fact, viz.: that in early age the large majority of cases, rare though they be reputed to be, exhibit localizations in the heart. For instance, Picot finds these in 37 cases out of 47, Claisse in 14 out of 18. As far as my own experience goes, I can say that I look upon the absence of heart complication in rheumatism, at that age, as very exceptional. Further, I know cases in which endocarditis was for some time the only manifestation of the disease, this preceding all articular affections, and many writers on diseases of children have made the same observation. At the present time I am in attendance on an intelligent and delicate little boy of four years, who, with the exception of slight pain in the knee-joint through one or two days, which, but for the unusual care and attention of the family, might easily have been overlooked, has had no symptoms of rheumatism, except well-marked endocarditis, resulting in mitral incompetency. The large number of cases in which the final results of endocarditis—venous stagnation, bronchial catarrh, epistaxis, chorea, dropsy—are the first subjects of complaint, bear out this observation to its full extent. In many of these the history of the first rheumatic affection will be remembered, in others it has never been noticed, or has been forgotten. This may happen so much the more frequently, as endocarditis itself may set in without much fever, or other symptoms; a fact which ought to be carefully kept in mind, and induce us to examine day after day, even the slightest case of joint affection, for its heart complication. Murmurs, in the beginning, when indicating serious lesions, will be accompanied with more or less rise of temperature. But when they are the result of mere congestion of the surface, and functional incompetency of the valves, no variation of the thermometer may be observed. Such a condition exists as well on the endocardium, as on the intima of the synovial membranes. Or a murmur may be a temporary symptom of irregular contraction of the heart, the consequence of a true muscular rheumatism of its walls. For this, although rare, may coincide with acute articular rheumatism. In both of these instances the murmur is likely to disappear, after some time, in the same manner as it will cease with the cessation of functional anæmia in advanced age, or even after certain slight inflammations occurring in febrile diseases, as variola. In children, however, anæmic murmurs are very rare indeed. Yet the diagnosis of endocarditis is by no means beyond the reach of a doubt, for, although a genuine inflammatory murmur *may* disappear, the majority of temporary murmurs are of a non-inflammatory character.

As the frequency of endocarditis in the rheumatism of infants and children is indubitable, is there an explanation of the fact? I look for it first, in the anatomical and physiological peculiarities of the young heart. In the new-born child the heart weighs from eight to thirteen drachms, while that of the adult weighs eight ounces. Thus the adult heart has but six times the weight of that of the newly born. But the weight of the whole adult body is twenty-five times that of the newly born. Thus, the newly-born heart is four times as heavy, in proportion, as that of the adult. With this relative increase in muscular substance, increased activity of its function goes hand in hand. Again, the manner of circulation of the blood-current must be emphasized. The circulation of blood, even in the large arteries, does not depend solely upon the action of the heart-muscle and the elasticity of the artery walls, but also upon the activity of the voluntary muscles. When these contract, the small blood-vessels are emptied; when they relax, these are filled by aspiration. This important factor contributing to rapid circulation is not so active in the infant, where the muscular system is but inadequately developed. Thus, the heart-muscle has to perform part of the labor which in advanced years is borne by the voluntary muscles. Nor is this all. From its ventricular orifice to the insertion of the ductus arteriosus Botalli the aorta is narrow in early age ; the young heart has to overcome this narrowness, in fact, has to dilate the aorta to its later norm. With its labor corresponds its size, as the size of a voluntary muscle with its exertion. Thus, the heart of a baby of fifteen months is about as large as that of a child of five years. But not only will its size grow with its labor, but also its danger. Any organ with a rapid physiological action—be this action nutritive, that of growth, or dynamic exertion—is liable to become the seat of pathological changes. A fine illustration of this point is yielded by the statistics of heart diseases in the fœtal and postnatal periods. Before birth, the function of the heart is principally performed by its right half, after birth by the left. In consequence, the diseases of the fœtal heart are met with on the right side in ninety or ninety-five per cent. of all the cases, while the large majority of heart diseases after birth, in any period of life, are found on the left side.

While I laid these facts before you merely for the sake of proving that the danger of an organ grows with its work, and that the frequency of heart complications in general rheumatism of the young is the result of its physiological dignity and labor, they prove something besides. You have heard that the large number of heart diseases in the newly-born and very young infant are confined to the right side ; they are congenital. The large majority of heart diseases in the child of five years and upwards are found in the left side ; they are acquired. What does that mean? It means the statistical fact that the congenital heart-disease seldom lasts into childhood ; it destroys life. It means, further, that almost all the numerous heart diseases of childhood up to puberty do not date from birth, but are the result of the most common cause of cardiac disease—rheumatism. And with this consideration in view, I know you will never forget that the doctrine of the rare occurrence of rheumatism in the young is an illusion, and be prepared to look for and meet with this grave disorder in many instances.

The long list of manifestations of rheumatism in childhood is not yet closed, however. The anatomical equality of serous membranes, and the nature of the meninges of the cerebro-spinal cavities, renders the effusion into these parts an a priori probability. But theoretical conclusions are not required where facts are frequent. For no complex of organs is so liable to rheumatic disorders as those constituting the nervous system. Not even peripheric nerves appear to be exempt, since Rigal observed the occurrence of a severe neuralgia of both face and abdomen, in a boy of fourteen, before the joints became the seat of the manifestation of the disease.

The attention of authors has been principally directed to the brain and its meninges. Symptoms of both irritation and depression have been noted. Hyperæsthesia, contraction of the pupils, hallucinations, oppression, melancholia and physical diseases in general, and coma, have been met with in many instances. Where and as long as the symptoms of irritation prevailed, the prognosis was generally favorable; symptoms of depression, such as coma, were invariably considered fatal. For it is a peculiarity of rheumatism, that its cerebral manifestations are more steady, less changeable, than are the symptoms of a common non-rheumatic meningitis or encephalitis. In post-mortem examinations a number of anatomical lesions were found in both brain and meninges. Anæmia, hyperæmia, meningitis, with œdema into the arachnoid, with effusion into the space between dura mater and pia, fibrinous deposits, thickening and adhesions of the pia, dilatation of the sinuses, fatty degeneration of blood-vessels, encephalitis in its different stages, softening of the gray substance of the large ganglia, emboli, and apoplectic deposits have been found. Over this array of conditions I pass so cursorily because I mean to add, at once, an important statement, viz., that they are principally based upon observations made on the adult. As a general rule, the symptoms resulting from affections of the nervous centres differ greatly in the young and old. Where you have delirium in the adult, you have convulsions in the child. The symptoms enumerated before belong principally to the sensitive sphere ; the same effusions in the child affect the motory powers principally. Besides, there is a peculiarity in the rheumatism of the young already alluded to, which I think is mostly explained by the rapidity with which effusions take place in that period of life, viz., that fever is generally less in the rheumatism of the young, and consequently its anatomical results, of which I have spoken before, and which are very marked in the nervous system, are less pronounced. A further difference is this, that a fatal termination is less frequent in the young than in the adult. Thus, very few post-mortem examinations are on record. Such as are mentioned, however, and the nature of the disease, and its essential equality with the same affection in the adult, facilitate our conclusion that the local lesions must be of a similar character, though not of the same gravity.

I have said that the nervous disorder manifested in rheumatism is of the motory order. All of you have seen, in the course of your studies, a number of cases of St. Vitus' dance, or chorea minor. You remember that the principal symptom was the inability of the will to control the voluntary muscles, to adapt and coördinate them to a certain purpose. The children twist and twitch while sitting, contort their limbs, stumble in walking, stutter in speaking, and drop knife and fork, or use them inappropriately. This symptom is not developed at once. Now and then you learn a preliminary history—pain, restlessness, nervousness and disturbed sleep. The first appearance of the motor disorder is mostly observed in the right upper extremity; after a while the lower extremity of the same side participates, and the rest of the voluntary muscles follow suit. In a large number of cases the affection follows this course; it is apt to be unilateral in the beginning. The sphincters remain unaffected. Not always, however, is the affection so general; frequently but a few muscles of the face, or face and neck are disturbed. During sleep the contortions will stop. In some severe cases they may, however, continue. In these the contact with the bed, and involuntary contractions of the muscles suffice to result in choreic movements. Muscular efforts during dreams will have the same effect. Most patients are between five and fourteen years old. While, however, adults, such as pregnant women, are liable occasionally to chorea, the very young are by no means exempt. E. H. Richter reports a case of chorea in the newly born; at the age of a few months it has been observed a number of times, and I remember a few cases at two and three years. The majority of patients belong to the female sex; they are mostly anæmic and thin, seldom in previous good health. Complications with nervous disorders of different kinds are not unusual; some of them are of a hyperæsthetic, some of a paretic, or paralytic, character. Both physical and intellectual efforts result in speedy fatigue. The intellect is sometimes impaired. Neuralgias, especially of the intercostal nerves, with distinct points of Valleix, are not infrequent. In a few cases I have noticed herpes zoster. In others there is unilateral paresis or paralysis, either contemporaneous with, or subsequent to chorea; in others, epilepsy. Grisolle relates a case of chorea complicated with paraplegia, which terminated favorably in a few days; Trousseau, a similar one of chorea, paraplegia and rhachialgia; Picot, one of rheumatism, chorea, endocarditis and paraplegia.

Some of the patients have a hereditary tendency to nervous disorders. In their families runs insanity, epilepsy, hysteria or diabetes. Other etiological facts are, injuries, mental emotions, irritation, or exposure to sudden changes of temperature. Some cases are of reflex origin. Pharyngeal, intestinal and sexual irritations play a prominent part in the etiology of chorea. Chronic pharyngeal catarrh, through its irritation of the trigeminus nerve, is a frequent cause of local chorea confined to face and neck. But the presence of worms in the intestinal tract is not so frequent a cause of disturbance in the young, with us, as in Europe. The prevailing belief in their influence is communicated to us from transatlantic countries, where the food of the working classes is very coarse, and worms are more frequent. This influence is exaggerated, no doubt; but sexual irritation is probably not estimated at its full importance. Masturbation is too common a habit amongst little ones to be overlooked; and still I know that the fact of its frequent existence is not sufficiently appreciated by my professional brethren. Bad habits and wickedness on the part of nurses, vesical catarrh and gravel, narrow prepuce and accumulation of smegma around the glans penis, vaginal catarrh and oxyurides in the rectum are just as many determining causes. But the main cause of chorea is rheumatism. The connection between the two was known, amongst older writers, to Stoll and Bouteille, later to Copeland and Bright, until in 1850 Lee and Botrel, and in 1866–68, in the *Archives Générales*, Roger made chorea and rheumatism the subject of elaborate and successful treatises. For a long time it was assumed that chorea depended on rheumatism through the intermediate link of endocarditis. Now, it is true that endocarditis is found in chorea; thus, Ogle reports its existence ten times in sixteen fatal cases. But you have heard that endocarditis is but seldom absent in acute rheumatism of the young. Thus it appears, that chorea, endocarditis and polyarthritis are but the coördinate symptoms of one and the same affection. If acute rheumatism was the cause of endocarditis, and endocarditis or rheumatism the cause of chorea, we should always find those symptoms in the same order. The effusion of the joint would lead, endocarditis would follow, and chorea finish the series. It is, however, not so. You have heard that endocardical rheumatism may precede the inflammation of the joint; and in the same manner chorea may precede either endocarditis ar polyarthritis. In a boy of three years, I have observed general chorea four or five days before the slightest symptom of rheumatism was perceptible in the joints. When the joints became affected the choreic movements grew less. After a week the articular swelling receding, chorea became more prominent again. In this manner nerve and joint rheumatism alternated three times in the course of two months, until finally the case wound up with a mild endocarditis, terminating in insufficiency of the mitral valve.

There must be some lesion either in the organ of the will (Klebs), or the centre of coördination (Cyon), or some other part of the nervous centre which causes the peculiar symptoms of chorea. The readiness with which the majority of cases get well, either temporarily or permanently, appears to prove that in this majority of cases the anatomical change can certainly not be very great. But an alteration in the nutrition of the parts we shall have to assume, leaving out of sight the few reported cases of cerebral tubercle, hypertrophy of the odontoid process, cerebral hypertrophy, and softening, which resulted, amongst others, in choreic symptoms. The alterations effected by fever alone vary with the height of temperature. Any long-continued change in the blood-vessels must result in serious changes of nerve tissue, probably of an anæmic character. Whatever changes take place, are probably most perceptible on the left side of brain. For the left carotid is the more direct route to the brain; its size is greater, the nutrition of the left brain more active; consequently the right side of the body more thoroughly innervated. In the same manner, and according to the principle that pathological action is liable to be in proportion to the degree of physiological function, the left brain is the seat of pathological lesions. A lesion in the left side of the brain is the principal cause of aphasia (complicated with right hemiplegia). So chorea begins on the right side, and there also are found some of its complications, such as paralysis. Of all the lesions, from simple hyperæmia to inflammatory changes and embolic infarctuses, each can lead to chorea, and such cases as are observed with long duration and great severity, fatal termination, or complication with paralysis, belong to the latter class. As the majority of cases are, however, temporary and mild, it is to be assumed that slight nutritive changes in the nerve centres are frequently the only causes. Restitution of these to their normal condition, would, in a month or two, relieve chorea. Thus the ready action of arsenic in most cases would be explained by its effect on nutrition and assimilation in general.

Hitherto, I have spoken of chorea as principally connected with lesions, or nutritive disorders, within the cranial cavity. There is one form, however, which is by no means very rare, and still has not been described, as far as I know, as fully as it deserves, viz., that class which depends on hyperæmia or inflammation of the spinal meninges. Brown-Séquard observed, that dogs suffering from chorea would still remain choreic after the spinal cord had been separated by a cross-section in its upper part. Onimus and Legros have rendered dogs choreic by injuries to the posterior roots of cervical spinal nerves and posterior gray column of this part of the cord. They place the seat of chorea here. In my own experience; spinal meningitis of the upper part is a frequent cause of chorea. Fifteen years ago, I reported the case of a girl of nine years who was presented with a violent form of chorea, the contortions of which exceeded anything I had ever seen before. The attack had come on very suddenly ; the child had been perfectly well before. It struck me that the face was less affected than the rest of the body, and thus my attention was at once directed to the upper part of the spinal cord. Pressure on the cervical portion, especially the spinal processes was exceedingly painful, and increased the spasmodic actions. Was the pain simply neuralgic ? or was it inflammatory ?

The thermometer answered the question very readily: The temperature was 104, and remained in that vicinity for another day. Ice, ergot, with purgatives, relieved both the meningitis and chorea within a week, confirming the theory of the etiology by the result of the treatment. This case is by no means a solitary one. I have seen a number which were just as marked as this one, and a great many besides in which fever and the extent of the inflammation were not so well marked. One of this kind has been presented to you a fortnight ago ; you remember a choreic girl of nine years, in whom pain, on slight pressure upon the cervical portion of the vertebral column, was very perceptible. In this case also the thermometer had to decide the character of the pain, whether neuralgic or inflammatory, and the etiology of the disease. The temperature ranged from 101 to 102, on several days, without any other symptoms to explain this rise of the temperature but the spinal affection. Mark the slightness of the increase, in that case, and remember that in the large majority of cases of subacute spinal meningitis the thermometer does not rise so high. For that reason it is important to avoid every possible source of mistake in your measurement. We have, therefore, preferred to measure in the rectum, rather than the axilla.

The differential diagnosis of acute rheumatism is but rarely difficult, though many of the symptoms belonging to the joints are not well pronounced in the young. Still, mistakes are possible. Inflammation of the tendons, or the subcutaneous tissue in the neighborhood of a joint, may mislead the careless practitioner. Foreign bodies in the joint, contractions of tendons, ought not to deceive a careful observer. "Growing pains" are not infrequently inflammatory rheumatism, and many an endocarditis of later years may be traced back to the "growing pains" which are but dimly remembered. In many instances, however, they are but the expression and result of muscular fatigue. Thus, sensitiveness and pain are the results of a chemical change taking place in the muscular tissue, in which phosphate of potassa and lactic acid are accumulated through over-exertion. When those products and their elimination are proportionate, no pain is felt; when the former is increased while the latter is retarded, the result is evident. Therefore, not only physical over-exertion, but insufficient circulation also results in the sensation of painful exhaustion. The latter acts through its tardiness in relieving the tissue of its cast-off material, and thus you understand why "growing pains" (not rheumatic) are so often noticed in pale, anæmic children.

An occasional source of error may arise from swelling of a joint resulting from hemorrhage into its cavity, lesion characteristic of that singular congenital disease, hæmophilia. Some of you recollect a boy of five or six years presented a few days ago in my clinic at the College of Physicians and Surgeons. His history yielded nothing but this, that after he was six months old, a tendency to bleeding became apparent. A slight cut would not close, nose-bleeding was frequent, subcutaneous and cutaneous hemorrhages took place on the slightest provocation, and when we saw him there were blue, black, yellow, green discolorations on several parts of the body, viz., both forearms, glutæal regions, left scapula, left knee, etc. The left knee, besides being discolored, was considerably distended. The swelling had begun, two days previously, quite suddenly, without any fever, and at the commencement without pain. There was no fever when presented, but considerable pain both spontaneous and on pressure, the result of the sudden lesion of the synovial membranes. Look out, then, for enlargement and inflammation of the joint, in cases of purpura, scurvy, and hæmophilia. When you take the accompanying symptoms into due consideration, you ought not to be mistaken, however, in your diagnosis.

Not quite so easy, sometimes, will you find, at your first visit, the diagnosis from some affections of the bones. In infants and children, the bones, before their final ossification, which is not entirely completed before the twentieth year, are more succulent, softer, and endowed with a more extensive circulation than in advanced years. Osteomyelitis, however, can hardly be mistaken for joint disease, as it is confined to the diaphysis, and reaches the epiphysis only through participation in the morbid process of the periosteum. In these cases a serious suppuration extending up to the joint may complicate the diagnosis. But such affections are, happily, rare, except, perhaps, in syphilitic, tuberculous, or scorbutic individuals. But the very neighborhood of the joint, or rather the epiphysis of the bones, and the intermediate cartilages between epiphysis and diaphysis, are occasionally the seats of perplexing processes. You know there is always a lively physiological action going on at the boundary line between the two, which is restricted by increasing ossification only. This latter takes place in different bones at different periods of life. The direction of the nutritive arteries determine its period. Where they converge, as at the elbow joints, ossification takes place early ; where they diverge, later. Therefore the knee-joint is exposed to osteochondritis more than any other, as well for pathological reasons as for mechanical ones. The intermediate cartilage is very apt to be destroyed by an inflammatory process. Suppuration may take place, the epiphysis secede from the diaphysis, and the joint get filled with pus. Thus, many an acute case of arthritis may puzzle you as to its original source. In the hip-joint especially, the determination of an only or principal cause of coxitis, and its original seat, may be rather difficult. For at birth, the upper epiphysis of the os femoris comprehends head, neck, and both trochanters. A few years afterwards, by progressive ossification, the neck forms part of the diaphysis, while head and trochanter major constitute each a separate epiphysis. Finally, however, the trochanter also undergoes the process of ossification, and the head alone remains, for many years, in its epiphyseal condition.

In conclusion, I have to speak, from a diagnostic point of view, of disorders of a nervous character which are the more perplexing to many medical men, the more they have been accustomed to look upon nervous (hysterical) symptoms as the privilege of the adult female. But the male sex may become hysterical, and the child certainly will in many instances. Now, I cannot here go into the question to what extent hysteria is found in childhood. Remember but this, until we shall find an opportunity to return to the subject, that nervous symptoms of the most serious types are not excessively rare in children, and are more than simply indicative of what will occur in future. Well-developed neuroses of the motory, sensitive and vaso-motory nerves are by no means exceptional in childhood. A girl of about eight years was under my treatment for some time, for a neuralgia of the right ulnar nerve, without fever or spinal complication. After some time a moderate swelling of the subcutaneous tissue of the carpus, and in the neighborhood of the shoulder-joint, was added to her difficulties. They disappeared, to be replaced only by a very severe neuralgic pain of the toes of her right foot. Her sufferings were intense for a long time; they appeared to be mitigated when an œdematous swelling of her right foot made its appearance. A protracted tonic and galvanic treatment was required to restore her. Another girl of five years was presented for acute rheumatism of the right shoulder joint, which was said to have lasted several weeks, and to be very painful. There was excessive sensitiveness to the slightest touch, and some swelling. But it struck me that since the commencement of the attack neither the heart nor another joint were attacked, that there was no fever, that deep pressure produced no more pain than superficial, that the pain extended over the n. thoracicus longus as well as the shoulder and upper arm; and finally, that the swelling was not exactly in the shoulder-joint, but above, nearer to, and to the rear of the acromial end of the scapula. Thus my diagnosis was secured. I had to deal with a neuralgia of the cervical plexus, and not with rheumatism. Just at present I have under observation a boy of eight years, who has a slight mitral incompetency, contracted while suffering from chorea some years ago. Two months ago he was attacked with rheumatism of both wrists, knees and ankle-joints. A number of the joints of the feet took part in the process. There was moderate fever and distinct swelling of wrists, knees and ankles. After some weeks his fever was gone, and swelling very moderate indeed. Still his complaints grew no less; he was taken with sudden attacks of excessive pains, gave rise to screams and yells, commencing about dark, and lasting all night; was very sensitive even in daytime to the gentlest touch, and exhibited such a disproportion between his objective and subjective symptoms that my suspicion was directed to other quarters than before. Then I recollected that in periods of great mental anxiety, his father, many years ago, suffered from very severe and well-pronounced attacks of hysterical convulsions, and that his mother, a refined, intellectual and neurotic woman, while the subject of oöphoritis, had been disturbed by neuroses both peripheric (mostly neuralgic) and cerebral. My little patient had no more fever for some time; there was hardly any swelling left; he was quite comfortable at certain times, screamed fearfully—without tears—on the slightest touch on certain points, and got frequently quiet under protracted and deep pressure, particularly when his attention was diverted to other quarters. The pain was, and is, not confined to those points mostly sensitive in sciatica; in fact there is no pain about the hip-joints or the sciatic notches. A number of cutaneous branches of the crurals are affected, as also the ramifications of the synovial membranes. At the same time, neither heart nor spinal cord participates in the process. The inflammation of the joint has been the cause of irritation in the sensitive nerves of both synovial membranes and skin. In accordance with the diagnosis of neurosis (neuralgic only, no vaso-motor complication being present), the treatment has been changed long ago. Iron and galvanism, with roborant diet and warm bathing, are the remedies on which I am at present relying, with a satisfactory, though slow, result. In all these cases I have, while I related them to you, pointed out the pathognomonic symptoms of importance, and, therefore, do not repeat them. Nor can I, at this occasion, relate cases of the same nature, though with different symptoms. However, I desire to impress upon your minds again the variety of forms and seats of a neurosis of this kind. It may be mixed in character—motor, sensitive, and vaso-motor. Paralysis or paresis, neuralgia, local eruptions and redness in certain territories of blood-vessels, local chills, perspiration, œdema, are thus explained. If not so mixed, neuralgic only for instance, this neurosis is very puzzling in the selection of its locality. The styloid process, the internal condyle of the femur are pet places. But any cutaneous branch, no matter whether near a joint, or on the general surface, of the sciatic, crural, obturator, saphenus, tibial nerves may prove the source of annoyance and suffering. In the adult, the "spinal irritation" of the public (formerly of the profession also) is mostly but a cutaneous or meningeal neuralgia.

The indications for treatment of acute rheumatism vary according to the character of the affection and the affected locality. Inflamed joints must be rested, local and general heat reduced, hyperæmia removed, exudation and internal pressure diminished, and pain relieved. A few remarks will probably suffice to point out the means of obtaining the required results as far as possible.

The inflamed joint, or joints, can be rested on wire, wood, or plaster splints, lined with cotton. In what position? An experimental injection into the knee-joint of the dead body determines moderate flexion. (Bonnet.) Thus it appears that this slight curvature is the easiest position for the diseased joint; it is that in which the cavity is the largest. But when the disease appears to last long, another consideration comes up. For some time after recovery the joint will not be available when flexed, although the result may not at all be an anchylosis. Thus it depends on individual cases whether the joint is to be fastened in either extension or flexion.

Local and general depletion have been recommended. The latter diminishes the temperature, but does not prevent it from speedily rising again. Besides, it increases hydræmia and the amount of fibrine in the blood ; both of these conditions facilitate effusion. Thus, you will hardly meet with a case in which general depletion will appear required. Local depletion has no such lasting ill-effect. But still its indications are limited ; for there is but a limited vascular connection between skin and joint. Besides, the intima which is hyperæmic is separated from the surface by the mass of the synovial membrane, which has but a scanty supply of blood-vessels. Moreover, the vessels of the surface and the synovial intima belong sometimes to different vascular territories. On the knee-joint only matters are a little more favorable, and, therefore, now and then a small number of leeches will prove beneficial, at least temporarily.

A better effect on the dilated blood-vessels is observed by the application of ice. It contracts blood-vessels, reduces the temperature, and prevents effusion and suppuration. For all of these purposes it is more reliable than any other application. Thus we are, as for other beneficent innovations, under great obligation to Esmarch, who has introduced ice into the treatment of arthritis. It is indicated in the acute stage, where swelling is considerable, and temperature high. While, however, it renders the inflamed parts anæmic and prevents effusion from taking place, it also prevents absorption of the effusion. Thus, after the swelling and temperature have been reduced, the indication for ice has passed. Then it becomes necessary to increase vascular pressure and stimulate the lymphatics by warm applications. Poultices, warm water, cold applications which remain long enough to become warm, and warm baths take the place of ice.

Thus the internal pressure is relieved. Now and then, where this pressure is felt to a disagreeable degree, and the neighboring muscles are affected with reflex spasms, distraction of the joint, by extension, is recommended on the plan which is followed in chronic coxitis. In most cases, however, this method is inappropriate, because too painful. A frequent method consists in local derivation. For that purpose both vesicatories and tincture of iodine are recommended. The former may either be kept on until vesication has taken place, or removed when the skin has become hyperæmic, and their application repeated. The latter acts favorably by causing dilatation of external blood-vessels, over a number of joints, and by thus relieving the internal congestion. Theoretical reasoning, more than actual proof, relies on the presumed stimulation of the vaso-motor nerves of the interior by the external irritant. From these several points of view,-the use of stimulating embrocations may be considered. In chronic cases they may do good (friction only will oftentimes)—in acute cases they will prove injurious.

When, after the reduction of fever, a sufficient time has elapsed for us to believe that no further absorption of effusion will take place, or where a fibrinous exudation in the joint remains stationary, with or without the complication of subcutaneous œdema, gentle compression is required. Collodium, flannel bandages, cotton with linen bandages, elastic bandages, plaster of Paris will be found serviceable. Puncture of the joint cannot be objected to when the contents are purulent. Extravasations of blood contra-indicate puncture; and serous effusions will require it in those rare cases only in which the effusion is very copious, of very old date, and the synovial membranes greatly expanded and in an abnormal condition. In a few such cases I have opened the joint with a fine trocar, always taking care that neither the cartilage was hurt nor air admitted. Dieulafay's aspirator has been successfully used for this purpose.

In this connection I ought to speak also of the galvanic current as a means of producing absorption. Mild currents passing through the joints from one to three times daily, and from five to ten minutes, each time, have a beneficial effect. When obtained, this may perhaps be attributed to a tonic influence exerted upon the walls of the blood-vessels and lymphatics; in virtue of which the rate of circulation is increased.

Finally, the indication for directly relieving pain may require the application of chloroform, belladonna, opium, or veratria, according to circumstances, in lotions or ointments. Severe pain may necessitate a subcutaneous injection of morphia. Atropine has been used in the same way.

Hitherto, gentlemen, I have spoken of external applications only. Internal medication is resorted to upon the same indications which have been set forth. To relieve vascular pressure, aconite, digitalis, veratrum, colchicum, or quinia, are administered. Whichever you may select, do not forget that all of them require larger doses than the usual proportion-tables in your text-books on materia medica appear to justify, and further, that whatever effect is to be obtained, must be secured speedily. The inflammatory process is a very rapid one, and the prevention of its spreading and resulting in copious effusion is worth while accomplishing. On veratrum I rely where the reduction of the pulse is a principal object; aconite and digitalis are slower in their action, but may be continued for a longer period. The beneficial effect of these different cardiac sedatives is more perceptible in the quality of the pulse—which becomes softer—than upon its rapidity. The general rule is to push the dose until the pulse has fallen considerably, but not to the norm, then to maintain the dose for two or three days; then cautiously diminish. At any rise in the pulse, the dose must be increased, for a recrudescence of the articular affection is threatened. This rule holds especially for quinia. Veratrum is, in careless hands, the most dangerous of the cardiac sedatives, and cannot be handled so freely. The dose must often be diminished more rapidly, lest the vascular sedation become excessive.

The effects of quinia have been studied extensively in the last seven or eight years, and a vast amount of literature has been accumulated through the combined efforts of investigators of all countries. Although their results have been meagre, they are still positive enough to justify its intelligent administration. Several facts appear to be established: First, that in spite of Briquet's apparently conclusive results, quinia has no direct effect upon the nervous system, either cerebral or peripheric. No effect on the sympathetic and pneumogastric nerves has been produced, and the effects upon circulation are not brought about by any direct action of its own on the vaso-motor nerves or the cerebrum. For when the medulla is cut, quinia will reduce fever, although the connection between blood-vessels and brain is destroyed. Secondly, it reduces the amount of uric acid in the renal secretion, also the number of white corpuscles in the blood, and, when given in sufficient doses, depresses pulse and arterial pressure, and reduces the temperature. In frogs, it reduces reflex irritability also. Besides, it acts as an antifermentative by interfering with the chemical decomposition of animal material. Now the qualities enumerated above render quinia the principal antiphlogistic. Amongst the prominent symptoms of inflammation we count the increase of white blood-cells, the dilatation of blood-vessels, the slowness of local circulation, and the accumulation, stoppage, amœboid changes, and finally emigration of white blood-cells. (Serous effusion is a coördinate effect of the mechanical obstruction.) I can imagine that the increase of white blood-cells alone suffices to bring about all the other changes. Remember that in the capillaries circulation is two-fold: in the centre the rapid motion of the red blood-cells; along the wall the slow, easily retarded, pushing along of the leucocytes. The simple fact of their considerable increase obstructs circulation both of themselves and their comrades more advanced in the scale of animal perfection. They rest, crowd each other, become unwieldy, assume different amœboid shapes and motions, press on the thin walls of the capillaries, and force their way through the dilated interstices. The mechanical obstruction in and dilatation of the smallest vessels acts on those of larger size behind them, which, although of normal consistency and function, will also become dilated. Quinia, by reducing leucocytes permits the capillaries, which have no elasticity of their own, to resume their original size under the pressure of the outside tissue, and the larger blood-vessels, endowed with an elastic adventitia and muscular layer, to contract over the reduced sizes of their contents.

At all events, no matter whether this attempt at simplifying an apparently intricate subject coverst he whole ground or not—(I believe it does)—this fact is sure, that quinia has conquered its place at the head of the antiphlogistic remedies. Do not forget, however, that small doses have no such effect. When indicated at all it ought to be given in a dose of five grains, once, twice, or three times daily, to a child of one or two years. What I taught more than fifteen years ago—that quinia and vascular sedatives in general are tolerated and required by the young in apparently disproportionate doses, is acknowledged as correct by the theory and practice of a large portion of the profession at the present time. I will only add, that you ought to be certain of the solubility of your preparation. The sulphate ought to be avoided. Select the bisulphate, or better, the muriate, and never forget that the stomach absorbs less under the influence of a feverish condition. The question of subcutaneous administration of quinia in rheumatism, has, I believe, not yet been mooted. But it is as plausible as for intermittent fever. When the stomach rebels against the remedy, the rectum may take its place. But it will absorb nothing unless in solution.

The majority of the remedies which are recommended in apyretic rheumatism, are either absorbents or derivants. Iodide of potassium (or sodium) acts as a diuretic, and, furthermore, appears to restrain the transformation of cells into connective tissue. Thus, it is effectually used, as soon as the acute stage of rheumatism is stayed, in pleuritis, pneumonia, glandular affections, and inflammations of the connective tissue in general. As soon as "organization" of exudations has taken place—that is, as soon as hyperplastic connective tissue has resulted from the nutritive disorder, the remedy proves unavailing. You know, for instance, from what I formerly said about the difficulty of reducing the size of tonsils or lymphatic glands in a chronic condition of enlargement; that in such cases the knife has to take the place of internal remedies. Thus, what effect you hope to obtain from the administration of iodide of potassium, ought to be looked for soon. No matter whether the rheumatic manifestation takes place in the joint, heart, or nervous centre, the iodide ought to be given early, immediately after the fever has been subdued, in doses of from fifteen grains to a drachm or more, according to age. Whether colchicum has any effect besides being an arterial sedative, and acting upon the mucous membrane of the intestine, is doubtful. The same may be said of colchicine. In three or four daily doses of $\frac{1}{160}$th of a grain each, which may be gently increased from day to day, to a child of four or five years, it is apt to produce vomiting and diarrhœa, with occasional relief to the general symptoms. Alkaline salts have been praised for their effect upon neutralizing supposed acids contained in the blood. You have heard that this acid condition is doubtful, and know that the amount of uric acid in the urine of children, no matter whether rheumatic or not, is not large. If it is an object, however, to neutralize uric acid, you will perceive at once, that potassa salts are better suited than those of soda. The former combining more readily with uric acid than the latter, bitartrate of potassa, or citrate of potassa, would be preferable to Rochelle salts or Vichy water. Their principal effect is, probably, besides the increase of intestinal secretion, to be sought for in the larger amount of urine, the secretion of which increases with its alkaline condition.

The indications for therapeutical interference in the manifestations of rheumatism in heart, brain, and spinal cord, differ hardly from those in the joints. The application of ice, the use of digitalis, aconite, quinine, iodide of potassium, follows the general rules. Endocarditis, and particularly pericarditis requires the immediate and constant use of ice, which will prove the more beneficial, the nearer to the surface the affected locality. Hence its beautiful results in pleuritis and laryngitis. When the acute stage has passed by, you need not fear the use of warm bathing of about 90° in heart diseases any more than in other subacute or chronic inflammations. The atheromatous degeneration of advanced age may contraindicate them, but no excitement of the heart's action is to be feared in children, in whom atheromatous degeneration scarcely ever exists.

I shall conclude with a few remarks on the choreic manifestation of rheumatism. Of the large number of remedies which have been resorted to, I rely principally on arsenic. I have alluded to that before. Next in order I consider bromide of potassium ; last, nitrate of silver, or atropia. Rest is secured by chloral-hydrate, or large doses of bromide of potassium ; the muscular irritability soothed by subcutaneous injections of woorara. Very efficient in protracted and feverless cases, as also in chronic cases of rheumatism in general, is a daily bath containing from three to five ounces of the sulphide of potassium, and the galvanic current. In several instances a moderate current conducted through the whole length of the spine has moderated severe forms of chorea, after therapeutical agents have proved unsuccessful. Most of this may have been known to you. What is not so commonly known, is my treatment of those cases of acute chorea depending upon meningeal or medullary congestion or inflammation, of the pathology and diagnosis of which I have spoken before. Antifebriles, mild purgatives, ice, sometimes tincture of iodine, and principally ergot, have been relied upon in my own practice. I conclude with a single remark upon the dose of the latter. I am positive that its failures are mostly due to insufficiency of doses. For some observers in this city have acknowledged that, after experiencing many failures with small doses, they know of no remedy the effects of which are more reliable than ergot since they have increased the quantity of the drug. Less than half a drachm of Squibb's Fluid Extract I rarely give. I repeat this dose three or four times daily. A child of four or five years may take from two to four drachms daily, for many weeks in succession. Bad results I have never seen. With the exception of a few cases recorded in the journals, the stories of poisoning, epidemic or otherwise, acute or chronic, concern individuals or communities whose constitutions were previously broken down by long continued misery and starvation.

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ON

PNEUMO-THORAX.

BY

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GENTLEMEN :—Air in the pleural cavity, which is the meaning of the term pneumo-thorax, is, with very rare exceptions, derived through an aperture either in the walls of the chest, or in a lung. An exceptional instance is the derivation of air or gas from the alimentary canal, by means of an ulcerative communication with the œsophagus, or, possibly, with the stomach. A fæcal abscess communicating with the cæcum has been known to evacuate through the bronchial tubes. In this case, had not protective pleural adhesions existed, the abscess might have opened into the pleural cavity, and air or gas would then have been derived from the intestinal canal. It has been supposed that gas may be evolved by means of chemical changes in the liquid products of disease within the pleural cavity, or that it may be secreted by the pleura. Neither of these suppositions has been demonstrated, and they are by no means probable. I shall restrict myself in this lecture to pneumo-thorax resulting from a pulmonary aperture, that is, the air derived from a communication between the bronchial tubes and the cavity of the pleura. I shall not speak of pneumo-thorax caused by an opening through the walls of the chest, save in connection with the operation of thoracentesis. A communication with the bronchial tubes may, in some very rare instances, be caused by the discharge into the pleural cavity of the contents of a hydatid-cyst developed in the lung, or by rupture of the pleura by blood extravasated beneath it. Exclusive of these instances, pneumo-thorax is dependent on 1st, empyema; 2d, interstitial emphysema; 3d, circumscribed gangrene of lung, and 4th, phthisis. I shall treat of pneumo-thorax, in this lecture, as occurring in these four pathological connections.

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* The substance of this lecture was given in clinics at Bellevue Hospital, during the winter of 1874–75.

I. Pneumo-thorax in Cases of Empyema.

The opening in these cases has its point of departure within the pleural sac; that is, it is from without inward, whereas, in each of the other pathological connections, the opening is from within outward. Almost invariably, if not always, when thus pneumo-thorax is secondary to pleurisy, the pleuritic inflammation is suppurative. In simple pleurisy, the liquid in the pleural cavity being sero-lymph, there is no tendency to perforation of either the lung or the thoracic walls. I have reported two cases in which the chest on one side remained permanently filled with liquid effusion. In one case certainly, and in the other case probably, this condition had existed for many years. A large collection of pus in the pleural cavity, on the other hand, will, sooner or later, make its way through either the walls of the chest or the lung, and sometimes in both directions. The occurrence of perforation of the lung is denoted by the sudden expectoration of pus. Pus is expectorated in more or less abundance according to the size of the opening. It may pass through a free opening into the air tubes in such quantity and with such rapidity as to destroy life suddenly, by suffocation, an instance of which has come within my knowledge. A case has fallen under my observation in which all the pus contained in the pleural cavity was expectorated within a short period, and the recovery was speedy. In general, the result is otherwise. Although a large amount may be expectorated daily, the accumulation within the pleural cavity is but little, or not at all, diminished, the continued production of pus keeping pace with the amount which is raised. If expectoration be relied upon for the removal of the pus, the prognosis is extremely unfavorable; death may be expected after a prolonged period of progressive emaciation and exhaustion.

I pass at once to the important measure of treatment in these cases. It is the evacuation of the pus through an opening in the thoracic walls, and making the opening permanent, so as to secure drainage, which is rendered complete by daily washing out the pleural cavity. In illustration of this measure, I shall proceed to review the histories of two cases, now in hospital (Feb., 1875), which I have repeatedly brought before you.

The man named B., a laborer, aged thirty-three, was admitted into Bellevue Hospital in April, 1873. He had been ill for three months, and the symptoms, at the outset, denoted acute pleurisy. At the time of his admission, the physical signs showed considerable liquid in the right pleural cavity. Pneumo-thorax did not then exist. The latter was ascertained to have occurred on the twenty-second day after his admission. On the previous day he had expectorated over thirty ounces of pus. On the 6th of June following, eighteen ounces of pus were removed from the chest by aspiration. He came under my charge on the 1st of July. At this time he was extremely feeble and emaciated. The pulse varied from 110 to 120 per minute. The fingers were notably bulbous. There was flatness on percussion over the whole of the right side of the chest, with absence of respiration, vocal resonance, and fremitus. He expectorated daily pus in large quantities. He suffered much from dyspnœa. The axillary temperature varied from 101° to 103°. I resolved upon making a free opening into the chest, with no expectation of anything but relief of dyspnœa. The patient's condition seemed to be desperate, and I had in view only euthanasia. On the 3d of July the chest was opened in the fifth intercostal space on the axillary line, and sixty-eight ounces of creamy pus, without odor, escaped. On the next day fourteen ounces of pus were removed by means of a catheter introduced into the opening, and on the 5th of July about five ounces escaped in the same way. The pleural cavity was injected with tepid water. The patient was now quite comfortable. On the 7th of July, the chest not having been injected the previous day, thirty-two ounces of pus escaped. After this date the tent was removed from the opening daily, and the pleural cavity injected with tepid water, to which was added a little carbolic acid. On the 14th of July it was noted that the patient had gained daily. The aspect was improved. The appetite was good. The purulent expectoration had ceased, and all the symptoms denoted a very marked change for the better. At this time there was very little discharge through the opening. On the 18th of July, the opening, which had become quite small, was enlarged by a fresh incision, and the daily injections were continued with the discharge of only a small quantity of pus. On the 31st of July, there being no discharge of pus, the injections were discontinued, and the opening was allowed to close. At this date he weighed a hundred and twenty-seven pounds, and he had evidently gained much in weight. On the 13th of August his weight had increased to a hundred and thirty-nine pounds. He had gained in one week eleven pounds. On the 8th of September he weighed a hundred and forty-three pounds. The fingers were less bulbous. His expectoration was slight. His aspect was healthy, and he reported well enough to leave the hospital. There was slight dullness on percussion over the whole of the right side of the chest, with feeble respiratory murmur and some mucous râles at the base. This side was considerably contracted.

The recovery in this case (for it seemed proper to consider it such) was one of the most wonderful of the events in my clinical experience. It might be likened to a resurrection. On the 18th of September, 1873, I presented the patient at my hospital clinic, as having recovered from pneumo-pyo-thorax. He had the appearance of health. I had his photograph taken, and you can judge by it of his healthy aspect. He was to have left the hospital on the 20th of September. Had he done so, and, like many hospital patients, never again been heard of, I should have supposed that the recovery was permanent. On this date he complained of headache, and he was affected with urticaria. A purgative was prescribed by the house physician. On the next day, the bowels not having been moved, he was directed in the morning to take a powder of rhubarb and soda, and at night ten grains of calomel with a scruple of the bicarbonate of soda were given. These remedies failed to operate as a cathartic, but occasioned vomiting. On the 22d he was suddenly seized with great dyspnœa and pain in the chest, together with rapid and irregular action of the heart. These symptoms were relieved measurably by an opiate, and on the following day the signs of pleurisy with pneumo-thorax, affecting the right side, again appeared. There was still dyspnœa with rapid breathing and febrile movement. From this date to the last of February, 1874, the symptoms were noted almost daily, embracing pulse, temperature, etc., and at short intervals, also, the physical signs. In a short time he began again to expectorate pus, and there was a considerable accumulation of liquid within the pleural sac. In November he passed under the charge of my hospital colleague, Prof. Loomis. On the 26th of December, a free opening was again made, and thirty-six ounces of inodorous pus were discharged. The aperture was kept open and injections employed as before. On the 28th of February, 1874, there was a discharge of about two ounces of pus daily from the opening. The patient was feeble, keeping the bed. The pulse varied from 92 to 120, the respirations from 24 to 30, and the axillary temperature from 99° to 102.50°.

This patient is still in hospital (February, 1875), and I have recently brought him before you. His general condition is decidedly better than it was a year ago, but he still, for the most part, keeps his bed. The physical signs, as you will recollect, show a circumscribed empyema, pleuritic adhesions having taken place around this space, and the lung appearing to be healthy. The improvement, since the second perforation, has been extremely slow, but there seems to me to be good ground for expecting ultimate recovery. The history of this case appears to show that, after his recovery from empyema with pneumothorax, the old perforation reopened under violent acts of vomiting, and then occurred empyema as a result of the perforation. In the first instance the perforation was from without inward, but in the second from within outward. My account of this case, although I have abridged the history as much as possible, has occupied some time, but I think you will agree with me that it is a case of great interest.

It is a curious circumstance that the case just given and the case to be now given, occurred in this hospital at about the same time, and from that date, now nearly two years, these have been, so far as I know, the only cases of pneumo-thorax from perforation of lung incident to empyema.

The second patient, G. De C., Italian, gardener, aged forty-six, was admitted April 26th, 1873. His illness began about five months before his admission, and the symptoms at first denoted sub-acute pleurisy. He had kept the bed for nearly a month prior to his admission. He had had very little cough and expectoration. The signs on his admission showed considerable pleuritic effusion. On the 27th of May, twenty-three ounces of serous liquid were withdrawn by means of Dieulafoy's aspirator, under the direction of Prof. Loomis. On the 17th of May twenty-six ounces of serous liquid were withdrawn in the same manner. The signs of pneumo-thorax were first noted on the 5th of July. The patient came under my charge on the 1st of July. Prior to this date the results of repeated explorations were noted, and they showed only pleurisy. On the 15th of July he expectorated muco-pus in great abundance. On the 18th of July an exploration by means of a hypodermic syringe showed that the pleural cavity contained pus. On the 19th a free incision was made, and eighty-two ounces of fetid pus were discharged. The aperture was kept open, and injections of tepid water with a little carbolic acid were employed daily. At this time the patient was quite feeble, keeping, for the greater part of the day, the bed, the pulse varying from 92 to 102, and the axillary temperature from 99° to 101.50°. He progressively improved, gaining in weight and strength. On the 15th of September he was able to be up and out of doors. At the end of my service, March 1st, 1874, there was still a discharge of about two ounces of pus daily, sometimes fetid and sometimes inodorous, but he held his own as regards his general condition. You will recall the appearance of this patient, whom I have recently brought before you: He has still some purulent discharge from the opening into the chest, but his aspect is healthy, and he is but little under his standard of weight and strength.

II. Pneumo-thorax in Cases of Interstitial Emphysema.

When we consider that it is not extremely uncommon, in post-mortem examinations, to find air within a circumscribed space in the sub-pleural areolar tissue, the air separating the pleura from its attachments, and forming bleb-like elevations or even tumors of large size, the infrequency of pneumo-thorax arising from the rupture of these, seems surprising. This etiology of pneumo-thorax is in fact inferential; that is, it has not been demonstrated for the reason that the affection as thus produced does not prove fatal. Prof. W.

T. Gairdner, in his work on clinical medicine, published in 1862, gives an account of a case communicated by Dr. Thorburn to the *British Medical Journal* in 1860, in which symptoms of perforation, followed by the signs of pneumo-thorax, were unattended by febrile movement or any symptom of gravity, and complete recovery took place after some weeks. The attack occurred at night, without any apparent exciting cause, but the patient for three days previously had indulged freely in rowing. There was no evidence of phthisis either before or afterward. The case was seen by Prof. Gairdner, who supposes that, at a point where the lung was weakened by some cause, there occurred a *pin-hole* perforation from rupture.

I have reported a case very similar to this, with the additional interesting fact that after the pneumo-thorax had entirely disappeared, perforation occurred a second time, ending in permanent recovery. In my case the patient, a peddler, aged twenty-nine, was seized suddenly with a sharp pain while carrying his pack. The pain soon disappeared, but he was unable to take any active exercise from want of breath. There were no other symptoms of importance. The left side of the chest was dilated; the intercostal depressions were obliterated; the resonance on this side was purely tympanitic; the heart was dislocated to the right of the sternum; there was no vesicular murmur, but auscultation furnished amphoric sounds and metallic tinkling. There was no evidence of the presence of liquid. In less than a month from the date of the attack, all the signs of pneumo-thorax had disappeared; the vesicular murmur was everywhere appreciable over the left side, and the heart had returned to its normal situation. He left the hospital and returned to peddling, carrying his pack on his back as before. After a few weeks he came to consult me for a return of dyspnœa. With this exception he was well. He now presented, as before, the signs of pneumo-thorax, excepting the amphoric sounds. I saw him again eleven months afterward. He was then free from pneumo-thorax, and in all respects well. He had given up peddling, and had become a farm laborer. You will find further details of this case in my work on the "Principles and Practice of Medicine."

The case reported by Thorburn is characterized by him, and by Gairdner, as unique, and, with the exception of my case, I am unable to cite one of a similar character. It is, however, not improbable that such cases have repeatedly occurred, and the affection has been overlooked, or the physical signs not been correctly interpreted.

III. Pneumo-thorax in Cases of Circumscribed Gangrene of Lung.

I shall pass by, with a very brief notice, pneumo-thorax occurring in this pathological connection. Cases are rare, but several have come under my observation. Situated near the superficies of the lung, and the protection afforded by pleuritic adhesions wanting, the sloughing, gangrenous mass is discharged into the pleural cavity. Acute pleurisy with pneumo-thorax at once supervenes, and a speedily fatal termination is invariable. On opening the chest after death, the air which escapes is intensely fetid. The diagnosis is to be based on the signs and symptoms of pulmonary gangrene, preceding and accompanying the pneumo-thorax.

IV. Pneumo-thorax in Cases of Phthisis.

Pneumo-thorax resulting from perforation caused by the rupture of a tuberculous abscess or cavity into the pleural sac, is sufficiently familiar to every clinical observer of much experience. This hospital has furnished several examples during the present session. Out of nearly seven hundred recorded cases of phthisis, in the analytical study of which I am engaged, there are twenty-four in which pneumo-thorax was a complication—a proportion of about three and a half per cent. The percentage is small, but phthisis is so common that the instances of perforation are not very infrequent. Certainly the instances would be frequent were it not for the pleuritic adhesions which, as a rule, occur early in phthisis; these are a conservative provision against perforation. The perforation in phthisis is always followed by pleurisy, with more or less liquid effusion, so that we have the conditions expressed by the term pneumo-hydrothorax. The liquid is generally serum more or less turbid with lymph; occasionally it is purulent, and sometimes fetid.

Inasmuch as the perforation is due to the bursting of either a cavity or a collection of liquefied exudation, pneumo-thorax does not occur in the early stage of phthisis. The accident, however, is by no means evidence of extensive pulmonary disease. The cavity or collection which bursts is sometimes small, and the tuberculous affection may be quite limited. This fact is to be borne in mind with reference to the possibility of effecting a cure. Indeed, as a rule, the greater the amount of phthisis, the less the liability to perforation, because protective pleuritic adhesions are more likely to exist in proportion as the tuberculous disease is large.

The occurrence of the perforation is generally denoted by lancinating pain followed by dyspnœa, the latter being intense in proportion to the suddenness and extent of the compression of the lung by air and liquid in the pleural cavity. When cases come under our observation in which pneumo-thorax has existed for some time, it is usually easy to fix the date of the perforation. This is not always so. In one of my recorded cases there had been no sudden attack of pain, and the dyspnœa had developed so slowly that it was impossible to determine when the accident occurred. In one case the date of the perforation could be fixed by the sudden pain followed by dyspnœa, but the patient, a laboring man, continued to work for several days, although with much difficulty. In most cases the dyspnœa becomes quickly intense, and, if the patient survive, after a few days the suffering from the want of breath diminishes, and it may cease entirely.

I do not propose in this lecture to enter into the clinical history of pneumo-thorax in cases of phthisis, but I wish to speak of an occasional event which has not been sufficiently noticed by medical writers. I refer to the filling of the pleural cavity with liquid. This sometimes happens. With the accumulation of liquid the air disappears, the lung is compressed into a solid mass, and the case is converted into one of either simple pleurisy or empyema. I have notes of two cases in which this occurred, the physical signs rendering unequivocal the diagnosis of pneumo-thorax prior to the filling of the pleural cavity with liquid. In these cases the dilatation of the chest with liquid, and the consequent dyspnœa led to the employment of thoracentesis. In one case the thoracentesis was by aspiration, no air entering through the opening into the chest; nevertheless, the signs of pneumo-thorax were immediately reproduced. A free incision into the chest was afterward made. In the other case a free incision was made without previous aspiration. In both cases great relief was obtained by the withdrawal of the liquid, but death took place within a short period.

I have notes of a case in which the pleural cavity was filled with liquid, and the previous existence of pneumo-thorax was not known. Thoracentesis by aspiration was employed in this case, and, after a week, pneumo-thorax, from perforation of lung, occurred. I am sure that other similar instances have come under my observation, of which I have not preserved notes. In the case noted, the patient was much relieved by the removal of the liquid, but death occurred soon afterward. In this case, in addition to tuberculous disease of the lungs, there existed pericarditis, peritonitis, and disease of the kidneys. In a case in which pneumo-thorax from perforation follows aspiration, it may be a question whether a perforation had existed, which had been temporarily closed by the compression of lung, and which is reopened by the expansion of the lung after the removal of the liquid, or whether the perforation first occurred after, and as a result of, the thoracentesis. That the latter may be true of some cases, seems to me not improbable. I can understand that the rupture of the walls of a cavity situated near the surface of the lung may be caused by the rapid expansion of the lung when the liquid is suddenly withdrawn, for, if the lung expand under these circumstances, the protective pleuritic adhesions are wanting. Moreover, if one of Dieulafoy's needles is used in aspiration, I can understand that the lung, expanding with the acts of breathing and coughing during the operation, and coming into contact with the sharp point of the needle, the pleura may be torn, and an opening made in this way, resulting in pneumo-thorax. I have no clinical facts showing the production of pneumo-thorax by either of these modes. But I wish to impress upon you the risk, rationally considered, attending the use of Dieulafoy's needles. In my judgment, the liquid should be withdrawn from the chest always by means of either a canula or a catheter.

An important practical question relates to the employment of aspiration in the cases in which it is known that pneumo-thorax preceded the filling of the pleural cavity. Is it advisable, under these circumstances, to withdraw the liquid? An objection to it is this: the compression of the lung by the liquid closes the opening, and, if this compression be maintained, the opening may become permanently closed. Another objection may be raised, namely, the rapid expansion of the lung may lead to a reopening of the perforation, when, if the expansion were to take place slowly, as would be the case if the liquid were removed by absorption, this result would not be so likely to follow. In a rational point of view, these seem to me valid objections, and I would meet them as follows: If the pleural cavity become filled after pneumo-thorax has been known to exist, I would not resort to aspiration so long as the quantity of liquid was not large enough to occasion suffering from dyspnœa, that is, assuming that the liquid is not pus—and this is readily ascertained by exploring with the hypodermic syringe—but, if there be sufficient dilatation of the chest to occasion dyspnœa, I would withdraw a certain quantity of the liquid, enough to relieve dyspnœa, leaving sufficient to secure the possible advantage of compression ; and the withdrawal of liquid within this limit may be repeated *pro re natâ.*

I proceed now to make some remarks on thoracentesis in pneumo-thorax, irrespective of the cases just referred to. Puncturing the chest to give exit either to air, or air and liquid, whenever the suffering from dyspnœa, due to dilatation, is great, is undoubtedly judicious as a merely palliative measure. This I have done repeatedly. But the inquiry has arisen in my mind whether it may not be possible, in some rare cases, to accomplish something beyond a temporary relief, by making a free opening into the chest, as in cases of pneumo-thorax incident to empyema. Let us suppose a case of pneumo-thorax from the bursting of a tuberculous cavity, the amount of phthisis small, the disease non-progressive, and all the circumstances favorable for arrest and recovery, aside from the perforation of lung. There are such cases, albeit they are infrequent. May we not hope that by a free incision the cure of pneumo-thorax is possible in these cases? The answer to this question must be based on clinical facts which are yet to be acquired. Meanwhile, I can see no objection to making trial of this measure. Pneumo-thorax occurring as a complication of phthisis is almost hopeless. In the majority of cases this complication destroys life within a short period. We may say that the prognosis involves only a question of tolerance. It is probable that a free opening into the chest will not shorten the duration of life, and it certainly affords great relief. It is therefore warrantable. I have recently in two instances acted in accordance with this view. In a hospital case I had a free incision made, after aspiration had been once employed, and the pleural cavity was daily injected. This case offered nothing in the way of a favorable prognosis, as the patient was greatly reduced before the occurrence of pneumo-thorax. He died twelve days after the operation; but he was free from suffering, the death being by asthenia. The other case seemed to offer a better prospect, as the patient was able to be up and out of doors when the perforation occurred. I saw him seven days after its occurrence. The affected side of the chest was largely dilated, and the suffering from dyspnœa was very great, obliging him to keep constantly the sitting posture. I suggested a free incision, which was made at once, and the patient remained afterward free from suffering; but death took place in eleven days. I need not say that these two cases do not give encouragement for an expectation of permanent benefit from the operation. I feel sure, however, that in neither case was life shortened, if it were not prolonged, and it certainly contributed to euthanasia. I cite the cases simply with reference to the warrantableness of the operation. It remains to be seen hereafter if, in any instances, it may do more than secure relief and prolong life.*

The duration of life, after perforation of the lung in cases of phthisis, is variable. In the twenty-four cases embraced in the collection of cases of phthisis which I have now under analytical study, the longest duration was ten months. I have, however, known this complication to be tolerated for a much longer period. The most remarkable instance of toleration with which I am acquainted came under the observation of Dr. I. H. H. Burge, of Brooklyn. I have elsewhere reported this case, but it does not seem to have attracted the attention which it claims, and I shall therefore read the account contained in my work on the "Principles and Practice of Medicine."

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* The "Transactions of the Society of Alumni of the College of Physicians and Surgeons," published in October, 1842, contains the report, by my colleague, Professor Lewis A. Sayre, of a case of empyema, in which a free incision into the chest was followed by recovery. In connection with that case, thirty-two years ago, Professor Sayre raised the inquiry, "in the empyema of a tuberculous patient, from the rupture of an abscess into the pleura, would we not be justified in tapping as soon as discovered?"

"The patient, aged eighteen, an accountant, was seized, four days before his death, with pneumonia affecting the lower lobe of the right lung. At the time of the attack he considered himself in good health. He was attended by Dr. Burge, who found the physical signs denoting pneumonia, and the affected lobe was found after death in the second stage of this disease. On an examination of the chest, in addition to the pneumonia, Dr. Burge discovered the signs of pneumo-hydrothorax affecting the left side. Metallic tinkling, with amphoric respiration and voice, were finely marked. The upper portion of the chest on this side yielded a tympanitic, and the lower portion a dull resonance on percussion. On inquiry as to the previous health, the patient stated that he was quite well when attacked with the acute disease ; that is, four days before his death. On close questioning, however, it was ascertained that he had had a slight cough for a year, but so slight that he thought nothing of it. He was not conscious of any deficiency of breath, was not subject to pain in the side, and, in short, there were no symptoms pointing to so grave an affection as pleurisy with pneumo-thorax. The left lung was completely condensed from compression. The lower lobe was readily inflated ; the upper lobe but slightly, owing to the free escape of air through a perforation as large as a crow's quill, situated on the anterior aspect of this lobe, about midway from the upper to the lower extremity. On closing the aperture this lobe was readily inflated. The perforation was gaping, the orifice being surrounded by a cartilaginous rim. On passing a probe into the aperture, it entered a small cavity of about the size of an American walnut. The cavity was smooth and lined by a membrane. This was the only cavity in the left lung, and there were no tuberculous masses. The whole surface of the lung was covered with lymph, dense and

and successive splashing produced within the stomach, pneumo-thorax is simulated. An error of diagnosis is to be avoided by finding the evidence afforded by the voice (bronchophony or increased vocal resonance and fremitus) that the lung extends to the base of the chest. Moreover, a circumscribed pneumo-thorax, situated in the anterior portion of the chest on the left side, is likely to cause more or less displacement of the heart.

A case of circumscribed pneumo-thorax, recently in hospital, was of interest as illustrating the physical signs, and also as showing tolerance and progressive improvement without thoracentesis. On admission, the signs of pneumo-thorax were limited to the anterior aspect of the left side of the chest, the respiratory murmur and vocal resonance on the posterior and lateral aspects showing that the lung was adherent from the summit to the base. Over the anterior aspect, the intercostal depressions were lost; the respiration and voice were amphoric; there was metallic tinkling, and succussion produced distinct splashing. The case served to illustrate these signs to a class engaged in the practical study of physical diagnosis. The affection did not give rise to great want of breath even on considerable exertion; the patient was able to walk up several flights of stairs to the amphitheater without much difficulty, and his condition as regards appetite, digestion, nutrition and muscular strength, was good. He entered the hospital early in December. In the middle of February the amphoric breathing had disappeared; metallic tinkling ceased, and it was difficult to obtain a succussion-sound; but the resonance over most of the anterior aspect was still tympanitic, with absence of respiratory murmur and vocal resonance. The embarrassment of breathing on exercise was now so little, and the general condition was so good, that he reported well enough to leave the hospital, and he was accordingly discharged.

It would have been of much interest to have had this case longer under observation in order to have ascertained whether the improvement, as denoted by the physical signs, continued to be progressive. The question arises whether, in a case like this, recovery may not take place without local interference. And, another inquiry arises, with regard to the treatment, namely, if, in a case like this, progressive improvement does not take place, would a free opening into the chest be advisable, or would it be better to hope for an indefinite tolerance of the affection, and not interfere. With reference to this inquiry, I would say, should experience show that making a free opening is judicious when the pleural cavity is filled with air and liquid, it is a reasonable inference that this measure is appropriate, under the circumstances stated, in circumscribed pneumo-thorax. I would answer the inquiry in this way provisionally, in the absence of data to be derived from experience.

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REST IN NERVOUS DISEASE:

ITS USE AND ABUSE.*

BY

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You, gentlemen, who have so often heard me dwell on the value of rest in certain diseases, and have seen how sternly in others I have urged the weary patient to move about, and to forsake the bed—you, I say, will not feel surprised that I should wish to group together the thoughts, maxims, and advice in which, from time to time, I have sought to convey my ideas on this fertile subject.

I am the more anxious to do this because I have often been misunderstood in regard to it, and am quoted by men who have not known the details of our treatment as having won this or that triumph over disease by merely putting the patient in bed, while, as you well know, it is the way we deal with the case after we have made sure of rest that makes this same rest a help or a hurt.

Then, too, it is well to say something of it for younger men, to whom it is a great stumbling-block in early practice, for the reason that they often find it hard to make a patient submit to long repose in bed, and quite as hard sometimes to get another out of bed.

When there is to come of rest sudden ease to a pained limb or an aching back, you quickly enlist the patient on your side: but when you propose to any one, man or woman, who can still walk, however feebly, to go to bed for a month or two, you must be able to make him or her feel sure that it is the best or only way towards cure. And this asks that you, in turn, have a firm trust in your own judgment, and a strong will to follow out its decrees. You must have, in a word, what the French call "the courage of your opinions."

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* The substance of this lecture was delivered at different times at the Infirmary for Nervous Diseases of the Orthopœdic Hospital in Philadelphia.

Think, now, what it asks of your patient to yield to such advice, and to yield with that calmness of trustful belief which alone will secure the rest of mind we want no less than the rest of bone and muscle and nerve. Sometimes I wonder that we ever get from any human being such childlike obedience. Yet we do get it, even from men. As to women, for some reason they take more kindly to rest than do men, and will stay in bed, when once there, as long as you wish, and longer sometimes. Indeed, he who says to a woman, "You are ill. Remain in bed for a month"—takes on himself a grave duty, and may not have the luck to get her afoot again, which is a thing to be thought of when trying some of these perilous therapeutics on your future patients. This is one reason why, in any case, and most of all in a woman's, whether we shall say walk much, little, or none at all, is so grave a question. It seems to the young physician easy to say to a woman who has been in bed for a month, and is able enough to get up: "Now the time has come for you to leave your bed." He finds, perhaps, that the woman has gained a set belief that she cannot get up, and that to give back to her the assurance of her ableness to walk is no light or ready task.

But suppose that in one of the forms of diseases of which I shall soon speak you have made up your mind to use rest. It will be well then for your patient that you should have formed some clear idea as to what rest is—what it can do, what it cannot do, and how far it may give rise to evil or end in good.

Rest and unrest have had their days and fashions in medicine ; but be you sure that he who can tell when the one is wanted, and when the other, is a man who is a master in the ways of healing. Surgeons and doctors for a long while have been using rest as one means of curing disease. Not by any means all of them have distinct views as to what it is they do when they put at rest a limb or the whole body ; and yet this is what we most want to know. Unhappily, we lack as yet some of the factors needed to work out this hard equation ; and until these are given we must in part only guess at the physiological results of rest, for to-day no man can tell me fully what is the difference in the products of the life of a limb at positive rest and in active motion. In fact, most that we know on this matter is purely empirical, and is in the shape of coarse clinical results. Still, even these teach certain things which you will do well to bear in mind. First of all is the thought, which should be ever with us, that few medical means are without their evil side. In our efforts to help, we too often harm, and we must take prudent care always that, in causing the largest share of good, we give rise to the least amount of ill. The one goes with the other as surely as shadow with light. To no medical measure does this caution more apply than to the use of rest.

Let us take the simplest case—that which arises daily in the treatment of joint-troubles or broken bones. We put the limb in splints, and thus, for a time, check its power to move. The bone knits, or the joint gets well ; but the muscles waste, the skin dries, the nails may for a time cease to grow, nutrition is brought down, as an arithmetician would say, to its lowest terms, and when the bone or joint is well we have a limb which is in a state of disease. As concerns broken bones, the evil may be slight and easy of relief, if the surgeon will but remember that when joints are put at rest too long they soon fall a prey to a form of arthritis which is the more apt to be severe the older the patient is, and may be easily avoided by frequent motion of the joints, which, to be healthful, exact a certain share of daily movement. If, indeed, with perfect stillness of the fragments we could have the full life of a limb in action, I suspect that the cure of the break might be far more rapid.

What is true of the part is true of the whole. When we put the entire body at rest we create certain evils while doing some share of good, and it is therefore our part to use such means as shall, in every case, lessen and limit the ills we cannot wholly avoid. How to reach these ends I shall by and by state, but for a brief space I should like to dwell on some of the bad results which come of our efforts to reach through rest in bed, all the good which it can give us, and to these points I ask your thoughtful attention, because upon the care with which you meet and provide for them, depends the value which you will get out of this most potent means of treatment.

When you put patients in bed and forbid them to rise or to make use of their muscles, you at once lessen appetite, weaken, in many cases, digestion, constipate the bowels, and enfeeble circulation. To say how all this arises, would need, not a lecture, but a book, and I can only hint at what I might call headings for thought. Defect of circulation is the main business to think about. A man in bed has his heart-beats brought down in number and also in force. Then there is for him no longer the constant momentary pumping out of blood from active muscles, and these aids to the heart failing, the distant local circulations suffer, and the blood flows around the muscles and not through them, and the skin ceases to be flushed by exercise and becomes pale and shrunken. To be small one moment and large the next is a condition of health for the vessels, and this fails with the want of exercise, so that when a man lies in bed the vessels lose tone, and when he gets up of a sudden, this is seen in the way the blood column enlarges the lower vessels, and leaving the head, causes faintness. Of these well-known facts I remind you only that you may the more fully see why I dwell so much on the means which must be used with rest in order to take from its avoidable evils.

I was struck with the extent to which these evils may go, in the case of Mrs. P., æt. 52, who was, as you may remember, in Ward 2. She was brought here from Jersey, having been prone in bed fifteen years. I soon knew that she was free of disease, and had stayed in bed at first, because there was some lack of power and much pain on rising, and at last because she had the firm belief that she could not walk. After a week's massage I made her get up. I had won her full trust and she obeyed, or tried to obey me like a child. But she would faint and grow deadly pale even if seated a short time. The heart beats rose from 60 to 130, and grew feeble; the breath came fast, and she had to lie down at once. Her skin was dry, sallow, and bloodless; her muscles flabby, and when at last, after a fortnight more, I set her on her feet again, she had to endure for a time the most dreadful vertigo, and alarming palpitations of the heart, while her feet, in a few minutes of feeble walking, would swell so as to present the most strange appearance. By-and-by, all this went away, and in a month she could walk, sit up, sew, read, and, in a word, live like others. She went home a well-cured woman.

Think, then, when you put a person in bed that you are lessening the heart-beats some 20 a minute, nearly a third; that you are making the tardy blood to linger in the by-ways of the blood-round, for it has its by-ways; that rest prone, binds the bowels, and tends to destroy the desire to eat; and that muscles in rest too long get to be unhealthy and shrunken in substance. Bear these ills in mind, and be ready to meet them, and you will have answered the hard question of how to help by rest without hurt to the patient.

But what is it that rest does for your sick man? We have seen, in brief, how it hurts; how does it help? When first I came to ask myself this question, I found that though in many cases I was sure of its usefulness, I was by no means sure why it gave rise to such helpful results; nor am I now much better off in this matter.

as concerns some of the diseases in which I am most secure of aid from it.

As regards a broken limb, the simplest case of all, it is plain that we gain chiefly an absence of deformity, and that rest is not needed to get mere union, which would usually take place whether the limb moved or not; unless the motion was very frequent and very free. In animals, the bones knit without splints, but nature secures absence of much motion, early in the case, by punishing movement with pain.

For the many disorders in which I or others have used rest, we shall find a great number and variety of reasons why this means is good, and perhaps my best way to teach you how it helps, will be to take up in turn these forms of trouble, and state with each the reasons why it is bettered by rest. But before doing this, I should remind you that rest is a relative term, and that we cannot, or, at least, that we can rarely get entire repose, and that, in fact, arrest is not what we want. We can only slow the works, and not stop them. Then, also, I ask you to keep in mind that the rest I talk of to-day is mechanical rest—stillness; and that we often bring about partial physiological rest by drugs, as when we compel sleep; or reduce the number of heart-beats; or make one organ active in order to make less the task of another. A very fair sample of this is in the treatment of certain stomach troubles. Here, for instance, is one from my note-book, of this way of helping a gastric neuralgia. A well-built woman, æt. about thirty, some years ago came to me from the country with a history of long emotional trials, ending in an agony of pain every time she ate. The first mouthful swallowed began the torment. We ran through a host of drugs and diets, from raw soup to milk—alike in vain. Then, thinking she might have an ulcer, I gave simple emetics, and studied with care what came up, but all with no gain. So at last I said I will rest the stomach; I therefore gave pepsin, acids, and beef-soup together by the rectum, which was thus made into a stomach. Meanwhile, although she could walk about, I put her in bed, because it is easiest when prone to keep the enemas, and because at rest she would use up least tissue, a point of moment when using so unnatural a way of feeding. Six weeks of this cured her of what I think was a neuralgia brought on by the functional activity of the stomach. There was some over-sensitive region in the centres, which suddenly translated into the language of pain the impressions which came to it from the acting stomach. The stomach and the sensorium, or so much of it as is in relation with the stomach, were put in splints, if I may so speak, and by-and-by got back their normal tone.

Following out this train of thought, let us go on looking at the neuralgias, and see why rest helps some of them, as it surely does. There is now in the ward for women a German girl, who has a neuralgia of the lower branch of the right fifth nerve. It never troubles her much unless she eats, talks, or laughs; in other words, moves the jaw, though even this is not needed, because if I handle the right corner of the mouth, push, stretch, or pinch it, the pain comes on at once. Neither in eating was it at first the jaw-motion alone that ensured the coming of pain, for even the act of swallowing would do it. Two things cause it to appear: the functional use of the mouth and throat, and any motion of certain parts about the angle of the mouth, where the neurility is given in part by the terminal threads of the nerve which has the pain. We might say that motion alone was the pain cause, but in some cases laughing or crying—normal convulsions of the face do not arouse the pain, and only talking can do it; or, as in a case we saw here last year, only swallowing, or, perhaps, as in a third case, the functional activity of the stomach, although there was no dyspepsia, and the first cause of the neuralgia was a disease of the upper jaw-bone. From all which we learn a lesson useful in other nerve pains, but useless here, because people must eat and digest, and will talk and laugh; and our lesson is this, that many neuralgias wake into fresh torment, owing to mere motion of parts, sometimes not close to them, and also to the functional use of parts near them, or related to them.

I once treated a case of infra-maxillary neural pain by rest. I forbade laughing and talking, and gave only fluid food. These means made less the number of fits of pain, which were usually about fifty a day. On the first day of rest they became thirty-three, and in four days came down to eleven, but these were very bad, as if with lessening the number we had made greater the pain. I found in this case that rest only helped, but did not cure, nor is it ever much more than an aid to other means. Could we have put a stop to swallowing, no doubt we should have done still better, for the more often she swallowed, the worse grew the pain. Motion of the part, then, increases nerve pain, and motion or active function of parts far from it, may do the same. No one can yet say why this is as regards the fifth nerve. The motions we speak of do not all of them mechanically disturb the nerve. In many cases they cannot. No doubt there are close relations in the sensorium between the centres which get impressions of all kinds from face, and throat, and mouth, and stomach, and when one little centre becomes irritable from disease, or the steady acting of an outside cause of disorder, like a pinched nerve, it soon begins to feel morbidly, through other ganglia, the normal impressions which every functional act brings to them, and through them to it.

Now this is not an explanation ; it is only a way of stating the facts, but it is a way which perhaps may aid you to see them in a clearer light. Suppress, by rest, the number of normal excitations which reach the over-sensitive nerve-cells, and you suppress some of the causes of pain, because an excitable centre feels all things as pain, and grows more and more sensitive, like a man in anger whose wrath is fed by everything.

The use of these facts gets to be plainer in neuralgias of the limbs. Some of you will recall a case of neuralgia from a wound of the median nerve-branches in the hand, which you saw last year. The irritations lasted long, and were so constant that the whole of the sensory centres belonging to the arm got at last to be distressingly alive to outside impressions, so that the faint messages sent to the brain from the motions of distant muscles in the forearm and arm were all felt in the centres as pain. This patient spent her life in keeping the member at rest. At last, by a nerve-section, the cause of trouble was cut off, and the central nerve-cells by slow degrees ceased to feel as pain the thousand every-day impressions, which are all the time, and in health without our knowledge, passing from the outside tissues to the centres within. This will, I hope, make plain to you one reason why rest, utter stillness, by making fewer the irritations of the too sensitive centres, is good as an aid to treatment. Now and always you will do well to keep in mind that there are no nerves of pain, but that whether an impression from without is to be that of a hurt or not, depends first on the grade of impression—in health only severe injury causing pain ; second, on the state of the centre, which may be tuned up to feel a feather-touch as pain ; and perhaps, third, though this is less clear, upon the state of the carrier, the nerve-trunk. But whenever any morbid state increases the power to feel pain, no cause is more potent than unrest of the part ; no help towards cure more certain than rest.

As I have pointed out before, motion increases the pain of neuralgia because it puts the part in function, and the mode in which this cause of pain acts is not easy to see through ; but motion has grosser effects in its direct mechanical influence on diseased nerves.

I cannot recall ever having pointed out to you here any instance of this, but I have seen it often. In my book on nerve injuries, I describe a case of nerve-wound of the sciatic, in which the man was quite easy unless he stood up, when at once a violent pain darted down the nerve, and with a cry he fell on the floor. This poor fellow had a local neuritis at the exit-point of the sciatic nerve.

Now let us look at the case of C. P., in the male ward. He has a severe sciatica, with all the usual symptoms. When he stands on the well leg, he has little pain, but if he rests on the lame limb, he has pain at once, and this goes on getting worse. This must be due to the squeeze which the contracting muscles give to the tender nerve, and more at the sciatic notch and under the great gluteal muscle than elsewhere, for in the notch lie two muscles, the pyriformis and the gemellus superior, both of which swell out and harden as we stand. I saw, four years ago, the sciatic notch at the after-death examination of an old German woman, in whom pain on motion was the most striking fact of an old sciatica. In her case, as probably in many such cases, the nerve-sheath was swollen and full of serum, so that the nerve was, so to speak, crowded in its way through the foramen, which, of course, made the least added pressure sore to bear. I believe that more so-called sciaticas are really cases of neuritis than is usually thought to be the case, and when the nerve is inflamed, another factor comes in to make mischief. Healthy nerves are meant to stand a good deal of stretching and pressure before they suffer or lose function, and just how far this may go, I have experimentally shown ; but a nerve long inflamed becomes hard and inelastic, and I cannot help thinking this must help to make motion painful. I used to watch with interest last year a patient of my colleague's, Dr. Sinkler. He was in Ward No. 3, and had, we thought, a neuritis of the sciatic. When going up-stairs, he never used the lame leg to mount with, because this act tightened the nerve over the edge of the notch. If, when standing, I made him swing the thigh forward with the leg extended, he had great pain, and extreme backward motion in the same position also gave still more distress for like reasons. A mere glance at the relations of the nerve to the notch and to the pyriformis and gemellus superior muscles will make all this clear enough, and will show you why entire rest with the limb in a midway-position is of all the easiest. So true is it, that you will treat in vain old sciaticas while they are up and about. Whatever else you do, put your patient at rest, and let it be complete. In some cases I have seen very good results from the use of splints, or from so encasing the hip with plaster, or collodion strips, as to limit movement. I should like well enough to say something as to the other means of treating sciatica, and as to the steady use of ice-bags in old cases, but there are many good ways of helping this malady, though, as I have urged, no one of them will prosper while the sufferer is afoot.

There is another form of neural pain which is very rare, but is so good as showing the effect of motion, and the uses of rest, that I am tempted to refer to it before speaking of rest in graver disease. The action of writing brings on in a few people one of four states, a spastic condition, known as writer's cramp, loss of power, a choreoid disturbance, or, very rarely, pain. I saw some time since a case in which there had been loss of power and cure, then choreoid troubles and cure, and lastly, extreme pain on writing, not in the fingers, but in the back of the hand. At first it went away soon after the patient ceased to write, but by-and-by it stayed, and at last, when it grew severe, it was kept up by any motion of the fingers. I simply placed the hand in a sling, and soon saw it get well, though I was forced to forbid the use of the pen, and to advise change of business to some out-door work. In common writer's-cramp, rest is the only help, and I, for my part, have seen no good come of any other treatments. Let such as believe in them try them without rest, and one such cure will end my doubt.

I am more anxious to say a few words as to the use of rest in certain central maladies, and in some disorders to which women are subject, disorders which I can more readily describe than label, and which I often find it easier to treat with success than to paint with clinical clearness.

Let us take first one of these forms of disorder with which you are all familiar in this house, and which we have yet to see a failure to cure. The cases I speak of, some doctors like to call hysteria, but hysteria is the nosological limbo of all unnamed female maladies. It were as well called mysteria for all its name teaches us of the host of morbid states which are crowded within its hazy boundaries. But whether or not the condition known by this much abused name be present or not in the cases I am about to speak of, this at least is clear, that it is not the key to their treatment, nor the main cause of them.

Let us take a case. Mrs. B. was brought to me on a couch from a distant New England State. She began adult life with certain over-work of the brain, a long, steady strain, backed by unusual mental force, and a certain vigor of will. Then she married early, and nursed in turn three children far beyond the common time of breast-feeding. Meanwhile, the claims of society, family, charity, and mental culture were all alike met, and at last, somewhat of a sudden, she gave way, lost flesh, came to weigh ninety-five pounds in place of one hundred and twenty-five pounds, or one hundred and thirty pounds, had pain in the back, steady dyspepsia, and great weakness. Everything tired her; to eat, to read, to digest, to move; and the least effort quickened her pulse, caused headache, and flushed cheeks, and at last pallor of face. There were no hysteric signs, but she had ceased to menstruate for at least three years, although I could find no womb-disease. Her look showed no anæmia able to explain her state, but the white corpuscles were one-third too many. The almost painful sense of tire was the main symptom, and it showed not only in what she said, but in her look of strange fatigue. Tied, as it were, to her chair or couch, she still strove to fill her place in life, but what it cost was seen in the lines of lassitude left on her face by this life of desperate effort.

As to treatment, she had tried everything, lived on tonics, and gone from doctor to doctor. When I saw her she could not walk more than a few steps, and her normal arm-pit temperature was but 96.5° to 97.5°.

It was clear to me that this woman, for reasons not plain, had never been able to get her tissues back to the standard of health. A twenty-mile walk would put your spinal ganglia in the state of exhaustion, which fell on hers after five minutes' use of her legs. Functional use of head or spine in health slowly and moderately flushes the part used, a simple provision for quick repair. But in this woman, the flush, I fancy, came almost at once, and the tire pain, which we all know, came also. The same remark applied to her mental efforts. Tire and pain went almost along with the functional action. Just, in a word, what you see in over-worked, busy, worried men was her state. Action *suddenly* dilated the vessels, and not slowly and in small degree. Over-distended vessels mean malnutrition, and so we have an endless series of evils, tied each to each in mischief-making sequence.

In her case I carried my treatment to an almost absurd extreme. She was put in bed, and left it for no purpose. At first she was even moved by her maid when she wanted to turn in bed. She was fed and washed by others, and forbidden to read or use her hands, and even to talk. Then she took iron as before freely, malt extract, a bottle a day, wine, raw beef, and plenty of butter, was fed, in fact, every two hours—at first little, then, after a while, more, and at first chiefly by the rectum, especially with cod oil.

But to be thus still, means, as I have said, feeble blood currents, stagnation in unused muscles, pale skin, disorder of the sluggish portal blood-round. And here is the gist of the treatment. You must get the effect of exercise without its ills. Exercise without exertion is what we want, and this was the way it was had. Every day she was masséed, thoroughly ; and skin, muscles, and belly kneaded until they flushed, and tingled with blood, and for the time rose in temperature two to four degrees. Every day each muscle was made to contract by faradic currents, and so she failed to feel the effect of disuse of the muscles. She was forbidden to exercise and yet had exercise, and she was fed largely but with watchful care.

At the thirtieth day she menstruated ; at the close of two months she had gained thirty pounds, and was at once able to walk upstairs to the attic, and down to the front-door untired, and in a month more, to go where she pleased, and do what she pleased. This sickly, feeble, wasted creature had become a handsome, wholesome, helpful woman, and so remains to this day, with only a constant gain in vigor.

These cases vary, of course, endlessly, but their essence is a state of reduced nutrition, which no mere tonic will cure while they are afoot and living on their capital. The main symptoms are the state of painful tire,* the low temperature, the great or less anæmia, the quick pulse, the excess of white blood. I have treated in this house, under the eyes of some of you, many such cases, in some instances blurred, as it were, by other symptoms, and sometimes in women whose every disease was painted on the puzzling background of hysteria. I should hesitate to say, as I do, that I have had no failure, if it were not that some of you have yourselves seen every case, and have wondered with me at the marvel of the coming back of life and bloom and power, and happiness.

The amount of feeding, of massage, and of faradic muscle exercise, which each case will bear and prosper under, is a matter to be told early in the case by watching the pulse, the temperature, and the appetite. In these cases the pulse is always rapid. If it fall, if the temperature rise, above all if there be the least gain in flesh, I know that I am on the right path, and am not moving on it too fast, but if these symptoms be reversed, and if the patient ceases to be hopeful, and looks weary, then, I lessen the passive exercise and wait a little, but above all I listen to what my masseur or masseuse tells me of the ease with which the limbs flush, of the readiness with which the muscles grow firm under the kneading fingers, for in this matter the rubbers get to have a very shrewd judgment. As to the rectal feeding, which I rarely omit, I say little, as it is now well understood. It should always include cod-liver oil. There is only this to be borne in mind : most medical men feed by the bowel when they cannot by the mouth ; I like to use both ends at once.

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* This symptom of ordinate sense of fatigue is found in many forms of disorder in women. The worst cases to handle are girls, with what is called spinal irritation by some, and spinal anæmia by others. The first name conveys to us nowadays, the idea of a group of symptoms — the second asserts a belief as to the cause of such symptoms. It has no clinical support. In these women there is at one time pallor of face, and the next minute islands of vasal dilatation. Perhaps the centres suffer like changes. I am not sure, and can offer as to this no other proof of value. It were easy to write long about this, and to be decoyed into more than a lecture.

It has long been my practice to insist that patients in the early stages of spinal congestion, meningitis, and chronic myelitis, especially in the very well localized forms the latter disease may assume, should lie down whenever they are in-doors. If I can carry my point, I like very much to put these cases in bed, and at perfect rest for a few weeks, and my reasons for doing so are these : Erect posture, and walking heavily, tax the spinal ganglia, and never more so than when every step is labor. The prone position puts them at rest. It is easiest to repair when the machine is not in full action, and so rest is to be wished for in a disordled spine. Besides which I could tell you case on case of these troubles, in which some sudden fatigue has brought about an abrupt increase in the gravity of the disease, while I have never seen ill come of rest even though it caused no distinct gain.

In cases of chronic meningitis, such as I have seen come of injury, or from the downward passage of cerebral meningitis after sunstroke, rest, with counter-irritants, is almost the only very good means; rest, long and complete, is the one utmost want. Nor need I say much as to the treatment by pure rest of the meningo-myelitis of Pott's disease, in which you have all seen the most perfect success in this house from the use of the prone position without the classical treatment by the hot iron, which is certainly sometimes useful, but in many cases needless, despite Charcot's recent and most authoritative statement.

I come last in my hasty review to a class of cases in which the use of rest is entirely my own idea, and in which I am sure I have gotten results of great value—results which happily you have seen with me in this very hospital. I allude to the use of rest in locomotor ataxia, in its early stages of pain chiefly and most surely, but also in any case of posterior sclerosis before it has gone too far. You will ask me what I mean by this last phrase, "gone too far;" and I answer that it is vague, because I really am, as yet, unable to set the limit beyond which rest is useless. I have seen it strangely relieve old cases of ataxia in which there was pain, and with the relief of pain there was also unhoped-for relief of other symptoms.

A number of years ago, a gentleman, æt. fifty, took a long sea-bath at Newport, and became thoroughly chilled. Next day he had a sharp attack of pain, here and there in the legs, like dagger stabs. A week later came another onset of like torment, and, after these, the attacks became common, so that at last no day went by without its share of pain. Two years passed before the ataxic symptoms grew into distinct form. Ten years after his first pain, while the constant subject of horrible torture, he fell in alighting from a street rail-car, and tore the internal lateral ligaments of the right knee. This injury kept him in bed three months, and during this time his pains grew fewer, and also less severe, and at last left him, to return no more, while also his ataxic symptoms were in a great measure changed for the better, and, indeed, so remained up to his death from pneumonia some years later. Five years ago one of my ataxic patients, who had also intense neuralgia, broke his leg, and, resting in bed for some weeks, left his pains between the sheets, as he said, and knew them no more.

Next I came to know of a gentleman who broke first his thigh and then his leg, and, resting in bed a long while, had also the good luck to get up cured of his ataxic neuralgia. A like case of one simple fracture with the same results came to my knowledge still later ; and by this time I began to see that rest—perfect, entire rest—had some curious power to aid the state of pain which makes one of the early, and often one of the constant distresses of ataxia.

Finally, in this very house, Mrs. B., a confirmed ataxic, with intense neuralgia, fell in walking, and broke her thigh, an event which put her in bed for six weeks.

She said to me one day : "It was worth while to break my leg, because now I have no pain." You will, some of you, recall her, as she was long an inmate of the women's ward. This case at length opened my eyes, and I felt ashamed not to have seen the full truth before.

In one of the male wards of this same hospital was a bad and very painful case of ataxia in the early stage. To test the correctness of my belief as to the value of absolute rest in relieving ataxial neuralgia, I kept him several weeks in bed. The result as to control of the pain was surely very remarkable. Before going to bed he could not walk without aid, nor could he stand for even a moment with closed eyes. The pain was inconstant, but never left him two days without extreme torment. Six weeks of almost absolute rest enabled him to stand a few moments with shut eyes, to walk unaided up and down the room, and to assure me of his entire freedom from pain since the seventh day in bed.

I do not think these cases can be looked upon as mere coincidences of pain ceasing about the time of the injury. I should rather conclude that exercise has power to flush the ganglia used in movement just as thinking brings blood to the brain and raises its temperature, and that this afflux of blood, or, at all events, the mere functional activity, is in some way injurious and irritating to the diseased centres. This will seem at least a reasonable view if we recall what I have said as to the influence of motion upon certain facial neuralgias. Even where there is no tender point, talking or chewing will often cause increase of pain, or awaken pain afresh. Thus, I have lately seen a case of frightful torment in the upper jaw, which was due to acid dyspepsia, and was cured when this state was relieved. The stomachal condition had created, however, a state of the nerve centres of the fifth nerve of such a character that if the patient attempted to talk or laugh, it presently resulted in a severe fit of pain, nor is this a very rare or merely curious example. Considering the spinal posterior ganglia and columns and the related sensorial centres above them, as in ataxia, ready to pass into the state which gives rise to pain, it seems likely enough that exercise may be efficient in bringing it on. Exercise does not only mean motion in a physiological view of its totality of results, but it also involves the passage centripetally of a host of impressions generated in the moving tissues, and of necessity passing up to the central sensory ganglia. The centres of motion and of sensation are, therefore, active during movement, and are then alike excited, so that we may, with these facts in view, see why motion may excite sensory organs.

It seems, then, that in the painful stage of locomotor ataxia motion is probably injurious, and that rest in bed is for like reasons useful.

In my original paper in the *American Journal of Medical Science* I stated that probably the bones of ataxics are brittle. I have since heard of a case in which both arms of an ataxic were broken after death while lifting his body to put it in the coffin ; and, quite lately, Charcot, in alluding to my paper, has given a number of cases which prove beyond a doubt that my suspicion as to the fragility of the bones of ataxics was correct.

You who have followed my clinics well know that I utterly disbelieve in the cure of posterior sclerosis. I have seen ataxic symptoms from spinal congestion and hysteria relieved, nay cured ; but I am sure that no one has yet seen a cure of ataxia from sclerosis of the posterior columns of the spinal marrow. Hence I venture to offer my treatment by rest as well worth a trial in the outbreak of the disease. Perhaps in the very earliest stages it might do more than merely relieve pain or stay for a time the unrelenting march of the disease.

During the war of the Rebellion, my friend Dr. Hammond, then the able and far-seeing Surgeon-General, placed under my care wards for nervous disease, in which I had the good fortune to see a host of strange and curious cases. Among them were some which are rarely met with in civil practice, although they are clinically represented by the cases of writer's-palsy, hammer-palsy, loss of power in the legs from use of the sewing-machine treadle, and the like. The cases I refer to were the most brilliant examples of treatment by rest that I have ever seen, and may fitly serve to close this lecture.

There were brought to us a number of these cases, which we soon learned to know almost at sight. They were men who had been over-marched—worn out by the use of their legs. Commonly these were people who had been scurvy-stricken, or victims of ague, and who, with vast force of will, had striven to keep up with retreating columns, or had, day after day, marched great distances, until at last they had literally given out, and been amazed to find that, on taking to bed, they were unable to rise again.

It was usually a pure exhaustion without sensory troubles, although a slight lack of feeling sometimes went with it. There were no bladder troubles as in congestion, and neither pain nor wasting, but simply loss of power.

We soon learned to treat these cases by rest in bed, with porter, beef soup, and strychnia. It was curious to see how promptly and completely they came up; and my colleagues, Drs. Morehouse and Keen, will still, I am sure, recall the amazement with which we saw their ready cures.

I have tried in this lecture to make clear my views as to the evil and the good of rest, but I have failed to satisfy myself if I have not set in strong light the fact that the ills which go with this useful means of treatment are capable of being met and dealt with to the good service of cases of disease. Rest can be made to help. Rest also can hurt, and he who deals with it as a means of cure must not fail to bear in mind the modes by which we can secure the light without the shadow, the good without the harm.

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SCIATICA.

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ALTHOUGH the term sciatica answers fairly enough to give a general idea of the affection which forms the subject of our present remarks, it can do no more. It would not be proper, for example, to define it as a disease of the sciatic nerve; for, in a considerable proportion of cases, the derangement may be limited to a few only of its remote branches, and, hence, may have as little connection with the main trunk as an overflow of the upper streams of the Ohio River has with the state of the Mississippi. Nor will it answer to define it either as an inflammatory or as a purely neuralgic affection; for there are cases in which there is neither a neuritis nor a perineuritis on the one hand, nor, on the other, any condition similar to those ill-understood neuroses in which a nerve-ache is present without an appreciable nerve-lesion to account for it; for the primary cause of the pain may, in some instances, be very appreciable, if not removable as well. Least of all can we give a definition based upon the etiology of the complaint, for the causes of sciatica are altogether too numerous to be crowded together under one category; the essential dissimilarity between some of the more common ones being so great, indeed, that it would seem to render doubtful the propriety of allowing any single term whatever to include them.

Nevertheless, clinically, it is not difficult to find good use for this term. Indeed, we could not do without it, for it would be useless to differentiate the various painful affections of the lower extremity usually classed together as cases of sciatica, into distinct diseases ; for we would not know where to stop in such a division. It is important, however, to direct attention at the outset to the variety of morbid states which this name is made to cover, in order to avoid the error of expecting, in this connection, that you are going to treat a disease instead of treating a patient. In three cases of luxation of the hip-joint, for example, you may properly think first of the rules for detecting, and then for remedying, such special accidents. In three cases of sciatica, on the other hand, you may have little more in common between them than pain in the leg.

In illustration of these remarks, I would briefly cite two cases in my experience, before passing to the history of the patients whom I now bring before you. The first was that of a gentleman, æt. forty-five, who had been suffering from phthisis of the right lung for over two years, and which had resulted in the formation of a large vomica, occupying the anterior region of the middle lobe. About the commencement of the summer season he began to suffer in his right leg from a pain which followed the course of the great sciatic nerve. This pain soon became terrific in its severity, but remittent, being worse in the afternoon and evening, and then declining towards morning, though never wholly ceasing. The paroxysms were of a deep, darting kind, shooting to the ankle, and sometimes to the dorsum of the foot and great toe. Constant tenderness was present in the popliteal space on pressure, and a steady gnawing or boring pain was complained of at the exit of the sciatic. During the attacks, the skin of the outer and posterior aspects of the thigh became exquisitely tender to the touch, and, lastly, numbness was felt by the patient, and loss of cutaneous sensibility could be made out by the usual tests, in various parts of the affected limb. Meantime his cough and expectoration almost wholly ceased. I treated him for about ten months with every measure I could devise, from subcutaneous injection of anodynes to repeated puncture of the sheath of the sciatic, and the most energetic applications of the iron at white heat—but all in vain. Finally, an elderly lady friend persuaded him to apply warm fomentations of sage leaves; and, from the first trial, he averred to me that he felt the pain assuaged, and in some ten days more it subsided almost entirely. I could only congratulate him on this efficacious [and] sage remedy; and also promised him that I would try it myself in the next case, before submitting others to the tortures which I had inflicted upon him. Unhappily, however, in a few weeks more he walked into my office to have me examine a large soft swelling in his right groin, which was divided by Poupart's ligament; and his subsequent story was that of a case of psoas abscess running its usual course. He never complained of a pain in the back until after the abscess had been discharging for several months; and, though I had long looked for some evidence of intra-pelvic pressure, yet, previous to the appearance of the swelling in the groin, he had no symptoms of the kind except the sciatic pain.

The next case was that of an unmarried woman, æt. forty, a laundress, who went out barefooted one wet day in spring to hang the clothes, and while doing so trod upon a piece of glass. This inflicted a wound between the first and second metatarsal bones of the left foot. The fragment of glass was extracted, and she put her foot in warm water, if I remember right, to allow the wound to bleed freely enough to "wash the poison out." From the moment that she received the injury, however, she felt a severe pain dart up the whole leg to the thigh, and she stated that that pain never left her from that time. She could not walk on the foot, and ere long the whole limb became stiff, cold, numb, and very painful on the slightest movement. After a while, acute darting pains began to be experienced, passing downwards instead of upwards, seeming to begin at the sciatic notch, then radiating over the buttock up to the top of the sacrum and the crest of the ilium, and also passing down to the knee and the external malleolus. The limb rapidly atrophied, was very cold, and the flexor muscles of the leg and the adductors of the thigh began to contract. From lying constantly on her back, also, on a hard bed, I found, on my first examination, a large bed-sore formed upon the sacrum. Patient experienced the most severe pain on pressure over the sciatic foramen, in the popliteal space, and under the external malleolus. The cutaneous sensibility, however, was decidedly diminished, as might be expected from the dusky, dry, and shrunken state of the skin. There were many other points of interest in this case, which we cannot allude to just now, though I will refer to it again as indicating the success of certain remedial measures in analogous conditions; but what I would remark here is, that, without the initial element in her history of a traumatic injury, she presented on my first examination nearly all the conditions usually regarded as characteristic accompaniments of severe sciatica; but, if so, they had their origin in a lesion at quite the opposite extremity of the nervous track from the preceding case.

The first instance of this affection, which I would take up now, is that of a patient whom I have shown to you several times during the past winter, in order to have you watch the progress of his symptoms and the effects of the treatment adopted. His history is as follows: J. P., æt. forty, single, native of Ireland, laborer. Family history unimportant; is a moderate whiskey-drinker; denies having ever contracted any venereal disease. Patient always enjoyed good health until he enlisted as a soldier in the civil war; and, in the year 1865, after exposure to cold and wet, he was attacked with intermittent fever, which lasted several months, and left him in a very debilitated condition. He had not recovered his former standard of good health up to the time when his present complaint began to manifest itself, which was just about two years before his admission into this hospital. It began with a sensation of pain and numbness in the left thigh and leg. The numbness soon passed off, but the pain persisted and increased in severity, coming on in agonizing paroxysms. The pain was of a sharp, lancinating character, commencing at a point above the sciatic foramen, and shooting down the posterior aspect of the thigh, around the knee and the back part of the leg. Patient could scarcely sleep from his pains, which, however, were not notably worse at night. He lost his appetite; was wholly incapacitated from work, and, as he expressed it, was "in every way used up." All movement of the leg was so painful that he had to keep it constantly rigid, and hence acquired a very limping gait. He soon noticed, also, that the affected limb was much colder than its fellow, and steadily atrophying. After wandering into various hospitals of several cities, with little or no improvement in his condition, he was admitted at Bellevue, Nov. 24, 1874.

On admission, patient's condition was generally reduced, with a countenance expressive of constant suffering from the pain, which, he states, has never been much relieved heretofore since it commenced. He, moreover, is further troubled now by a bad ulcer on the outer side of his leg, caused by an application of strong aqua ammonia. The ordinary functions of the body were normal, and he has always been free from hemorrhoids. Examination of the affected limb showed that it was both stiff and cold, and measured two and a half inches less in the circumference of the thigh at its middle, and an inch and a half less than the right in the calf. The great toe was rigidly extended, the heel raised, and the whole limb carried directly forward in walking, without flexion at the knee. Pressure at the sciatic foramen elicited immediate shooting pains down the whole posterior aspect of the thigh, and pains ascending upwards and diffused over the sacrum. Pressure in the popliteal space was equally painful, and radiated to the external malleolus ; while, midway down the calf, another tender spot caused pain to extend along the course of the anterior tibial nerve to the great toe.

We have, so far, several points in this case to notice. First, that the patient's constitution had become seriously deranged by malarial infection ; and this is the more worthy of consideration as it occurs so commonly in this country, as an element in the story of such patients, that it suggests inquiry as to a causal relationship between the derangements induced by ague and the occurrence of sciatica. My argument for the probability of such a relationship is this: First, that this great nerve of the lower extremity is far more apt to suffer from extensive vascular impediments than any other nerve of the body. In comparison with the sciatic nerve, the trigeminus, for instance, has scarcely any organs contiguous to its course, the state of whose circulation may influence its condition. But the portal vein may cause an indefinite number of mischiefs in the pelvis, according to the retardation in the current of its terminal branches, caused by a congested state of the liver. Constipation, hemorrhoids, perirectal inflammations, prostatitis, cystitis—and, in women, an unmentionable series of ailments—may all be traced, more or less, to the effects of portal obstruction. Moreover, the most famous mineral springs in all countries are those which, like the Congress of Saratoga, and the Kissingen of Germany, contain the chlorides of sodium and potassium, with the magnesian and lime salts in proportions very closely like their respective proportions in the serum of the human blood. Now, such medicinal waters have gained their reputation mainly from their efficacy in removing portal congestions, and thus become famous for curing hemorrhoids, bladder troubles, uterine disorders, and, lastly, sciatica—all by their property of exciting a natural elimination by excess of natural saline foods, and which elimination necessarily must unload the portal system. We shall soon show, also, that mere mechanical obstruction in the lower bowel has often proved to have a connection with the occurrence of sciatica, by the prompt cure of the nerve-ache after the dislodgement of the obstacle; and, hence, such facts, *a fortiori*, make the surmise highly probable that the far-reaching effects of portal congestion may induce sciatica in many ways. But, of all causes of portal congestion, in this country at least, ague is one of the commonest and most persistent. Liver and spleen alike seem to escape from the control of their vascular nerves almost as surely by the operation of this poison in weakening their vaso-motor ganglia, as the circulation of the face and head is unbalanced for years by the injury of a sunstroke. In every case of sciatica, therefore, I would recommend you carefully to investigate the condition of the portal circulation; and, if the patient admits a prolonged experience of troubles of malarial origin, that you pay good heed to this matter thereafter.

Another feature which I would allude to is the illustration which this case affords of the occasional annoyance you may experience from the application of strong counter-irritants to the skin of a limb affected with sciatica. The ulcer which was caused by the aqua ammonia used on him before his admission, was peculiarly painful and indisposed to heal. In the next case about to be related, a very moderate irritation from a bath of sulphuret of potassium caused several extensive and troublesome, though superficial, ulcerations; and I have just mentioned the large sacral bed-sore in the woman with the injury to the foot. The explanation of such mishaps is that the general nutrition of the limb, lowered both by deficient arterial circulation and by nerve perversion, as indicated by the resulting atrophy, will manifest an equal diminution in its reparative power against injuries. I have become, therefore, disinclined to use either blisters or issues in many of these cases, though counter-irritation is one of our most valuable remedial measures in their treatment; and I greatly prefer the quick and powerful impression of the actual cautery, provided always that the highest degree of heat possible can be employed. Iron or glass at a white heat act very differently, as regards both physical and remedial effects, from the same substances raised only to the point of red heat. At a white heat, the superficial layer of the epidermis is instantly shrivelled up, but the penetrating effect of the application is arrested by the same means which enabled Boussingault to show the wonder of making ice in a glowing crucible. The sore thus caused very rarely ulcerates, and the operation itself is much less painful than an ordinary burn or scald, while its specific remedial effects are quite distinct, and more certain than those which we get from the slower forms of counter-irritation. Glass has the advantage over iron that it is always smooth—and, what is more, can be quickly and hence conveniently heated to the requisite degree in the flame of an alcohol lamp. But, as it loses heat with equal rapidity, it cannot be used in the linear fashion, but must be applied by single touches. The plan, therefore, usually followed with it is to heat the tip of a glass pipette or small rod in the flame of the lamp (held in the left hand only a few inches from the patient's skin) ; and then, quickly turning the glowing point to the required spot, which has been previously indicated by a dot of ink, heating the glass anew for each application. If your applications should extend along the nape of the neck, or along the spine, for example, you make ten or more touches, about an inch apart, on either side of the spinous processes, and in a few minutes afterwards you will notice that the redness of the burns, instead of diffusing itself in all directions equally, will rather join in the line of the adjacent burns, and thus present the appearance of one continuous and narrow irritation.

Now, the treatment of this case and the results are in brief as follows : Nov. 24th. Ordered him to have a hypodermic injection, night and morning, of gt. x of Magendie's solution of morphia with the $\frac{1}{42}$d of a grain of sulphate of atropia, and to have the limb showered with hot and cold water alternately for fifteen minutes, three times a day ; or, rather, the douche with hot water was to be varied by a few sudden dashes of cold water.—Nov. 25th. Patient says he has less pain than yesterday, and can use his leg better. Ordered to-day to begin taking cod-liver oil and the Ward Mist. Ferri, and to have ten applications of the hot glass rod, each an inch apart, in the course of the sciatic nerve, commencing at its point of exit—the last two inches above the knee joint. Continue with injections and douches as before.—Nov. 26th. Patient endured the cauterization well, stating that he could stand anything which held out a chance for recovery, or even relief from pain.—Dec. 1st. Patient states that, up to last night, he had not had a particle of pain since the application of the cautery, but was awakened shortly after retiring by a return of pain, which prevented him from getting a wink of sleep for the remainder of the night. The reason for this relapse seemed to be that on receiving permission to go out of the hospital for a walk, he did so by taking a walk of over two miles on a rainy day; and though "not hurting him much then," it hurt him afterwards in bed. He had then an extra dose of morphia administered subcutaneously, and ten more applications of the glass rod were ordered, each point to correspond to a posterior sacral foramen.—Dec. 2. Patient permitted to keep his bed this morning, as his back is very stiff after the operation. The pain in the leg, however, was almost immediately relieved by it, and he passed the following night in comfort. The former burns are healing rapidly, and give him no inconvenience; while he feels well and expresses much satisfaction.—Dec. 5. Patient is up, and able to get about the ward with more facility than at any time since admission; and states that he is more free from pain and can walk better than at any period in eighteen months. Continue treatment, both medicinal and by douche and hypodermics. These have wrought a great improvement in his leg: the coldness of the limb has disappeared, the muscles are more supple and fuller, and he walks rapidly about the ward.—Dec. 20th. Up to the present time no repetition of the cautery has been required, as patient has had but one slight touch of pain, and that was three days since. The circumference of the left thigh has increased half an inch over the measurement of the same on admission.—Jan. 4, 1875. Continued improvement; heat and sensibility in the affected leg normal, and he has no pain. Burns on the sacrum almost healed.—Jan. 9th. Feeling very well; the left thigh has increased 1¼ inch and the calf ¾ inch since admission. Discharged to-day.—Jan. 24th. Patient paid a visit to the hospital to-day. States that he never felt better in his life; he has no pain whatever, and thinks that he is perfectly well.

This is, on the whole, the best case for results which we have to offer to-day; and the first element in his history which I would have you note now, is that of the coldness and atrophy of the limb, for a good part of the treatment was directed in the first instance to this condition. What caused the whole leg to lose heat and bulk? Not paralysis, for he never showed symptoms of either motor or sensor weakness, other than those which invariably accompany defective circulation in a part, with pain on movement. The coldness was not owing to obstruction in the venous circulation, for the leg was not swollen, but the opposite. Instead of that, both the atrophy and the defective temperature were due simply to a marked diminution in the supply of arterial blood throughout the whole limb—a conclusion which had its own corroboration present in a sensible difference in volume between the arterial pulse of the two lower extremities. But the arterial anæmia will also go far towards accounting for the other two features of the case; namely, stiffness or rigidity of the muscles and pain on using them; for one of the very first symptoms in a muscle that follows its deprivation of arterial blood by ligature or otherwise, is tonic and painful cramp. In those cases, also, for example, of sudden enfeeblement of the heart, occurring sometimes in the course of the exanthemata, or in pernicious intermittents, when the heart begins to beat faintly, and can propel the blood only into the neighboring great viscera, so that lungs, liver, spleen, kidneys, etc., become engorged, while the surface of the body turns white and shrunken, and the nurses are frightened, by the fading of the eruption, into the idea that the disease has "struck in ;" in such states one of the most distressing things to the patient is that the calves of the legs and the flexors of the arms are severely cramped. The cause of this symptom is that the arterial wave does not now reach those more distant parts, and they are for the time in a condition quite analogous to that present in rigor mortis ; and which cramp, in turn, speedily passes off, so soon as the heart regains its power. Let any cause, however, operate—not as in these instances, suddenly and transiently, but continuously, to lessen the arterial supply of a muscle, and the inevitable result will be, not only tonic contraction of the muscle, but its wasting as well. Now, it is difficult to examine a limb affected as this man's was, without concluding that the leading symptoms are due chiefly to some influence producing a steady contraction of the femoral artery itself, with all its branches ; and the observation of both physiologists and pathologists supply us with facts which strongly support this inference. Brown-Séquard found that irritation of the crural plexus in dogs produced so much contraction of the femoral artery that it was difficult to inject the artery of that side, while it was easy to do so in that of the opposite leg. On the other hand, in the days of frequent venesection, cases were constantly occurring where the lancet, by wounding some twig of the median nerve at the bend of the elbow, produced first a shooting pain up the arm, and sometimes up the neck ; and then this was in time followed by continuous pain in this direction, then by coldness of the arm ; and, finally, by rigidity of entire groups of muscles, if not all the muscles of the arm, with rapid and sometimes permanent wasting. An accident of this kind, doubtless, was the cause of the serious disabling of the leg in the case of the laundress above cited. Such facts all go to prove that an irritant sensor impression may be so reflected upon the vascular nerves of a part, that the arteries supplied by them may be kept in a state of permanent contraction, long enough to produce the most serious results, both as regards nutrition and function. Now, why should not a similar irritation produce similar effects, though there was no traumatic injury at the beginning? The causes which induce vaso-motor irritation are exceedingly numerous, and the nerve associations by which such irritations are often transmitted are, many of them, of great interest to study. Among these causes, cold is one of the commonest and most potent. A piece of ice in the palm of one hand will lower the temperature of the other palm as much as 15°, and effectually check the flow from a severed artery there. Cold to the feet contracts the arteries of the pelvic viscera to such a degree that an operation for stone may be rendered almost bloodless, in some cases, by putting the patient's feet in cold water. But what is more specially significant to our present purpose is that cold to the feet during menstruation not only shuts off the arterial determination to the uterus, but the irritation may be radiated from the ovarian plexus to the vascular nerves of the alimentary canal, producing long-standing anæmia of the intestines, with constipation from both tonic contraction and paralysis of the bowel, and, finally, general starvation. That the atrophy and pain, therefore, of many cases of sciatica are owing to vaso-motor irritation mainly, and not to mere affection of the sciatic trunk itself, has long been a conviction in my mind ; and I always commence the treatment of those cases where atrophy and rigidity are present, with measures directed towards remedying this supposed lesion.

To do this, we begin with attempts to blunt or paralyze the irritant sensor impression which is the starting point of the whole trouble ; for, so long as it persists, so long will it be reflected through the ganglia it reaches upon the motor filaments which proceed from them to regulate the calibre of arteries. It was, therefore, for this purpose, and not for curing his sciatica, that we ordered the subcutaneous injection of morphia and atropia together for this patient. After every such injection the limb would grow one or two degrees warmer, thus indicating a return, for the time being, of blood from corresponding relaxation of the arteries. But, by themselves, these neurotics would have proved totally inadequate ; for, like all other real neurotics, their effects are transient ; and hence, in a few hours, you will note the arteries contracting again, and the coldness and rigidity returning as before. I may say in this connection, however, that atropia has a greater control over tonic contraction of the unstriped muscular fibre, which lines the tubular structures of the body, and the cavities which are dilatations of those tubes, than morphia. The latter relieves pain better than atropia, but atropia relieves spasm of involuntary muscles, like those in the arterial walls, far more certainly than the opium alkaloid, and hence the advantage of combining the two in this present instance. Some authors are puzzled to know how these two agents can promote each other's operations when they are rightly reckoned as antidotal to each other in poisoning of brain centres by either. I have used belladonna to counteract opium poisoning in six cases, in private practice, and I know that it certainly can neutralize opium, apparently indefinitely, if employed in time, before secondary effects of opium are developed ; and my observations lead me not to care who contradicts this statement. But there need be no trouble whatever in your minds on this subject, if you remember that there are no agents at all which act on *the* nervous system. You hear of general nervous stimulants and of general nervous sedatives, and the like ; but the simple truth is that there is no "general nervous" anything, except death. The most extensively operating neurotics—like opium, for example—act only upon a few nervous ganglia or nerve functions, and leave thousands of other ganglia or nerve functions wholly untouched. Besides, opium acts on certain nerve ganglia in one way, and on other nerve centres in quite an opposite way, and that at the same moment; and so does atropia in its actions. Now, you can readily see, therefore, that there can be no such thing as a neurotic being a proper antidote to another neurotic, like chemical antidotes are in their operation —for all it can do will be to neutralize in some of its operations on certain centres or functions some of the operations of another neurotic on just those centres; while on other centres the actions of both may be so similar that they may virtually assist one another. Morphia and atropia, indeed, may be likened to a zealous Protestant forming a business partnership with an earnest Catholic—both ready to carry on mercantile enterprises together, and both equally ready to contradict one another in religious matters. It is difficult, indeed, to put up longer with the looseness of thought and language which speaks of the nervous system as a unit, or that it ever acts as a unit, or can ever be acted upon by any one agent in one general and universal way. It would be an extraordinary case of nervousness, indeed, if the entire nervous system could be stimulated, all the special senses together, with all the brain centres of thought and emotion, those that make the heart beat fast with those which make it beat slowly, the flexors and extensors of each limb simultaneously, and so on ad infinitum. Never answer, therefore, that any medicine "acts upon the nervous system," but only at most on a few of its centres or functions, and almost always very differently on different centres from the moment that it begins to act at all.

Much more effective, however, than any medicinal agent for paralyzing irritant, afferent, or sensor impressions is a remedy which can be obtained anywhere and to any desired extent, and that is simple warm water. The contact of water near the temperature of the blood which circulates about the cutaneous nerves, produces on those nerves a sedation almost, if not quite, equal to the effects of a general bleeding. A prolonged contact of warm water, indeed, will often so devitalize the nerves of a part that they can be roused into action thereafter only by strong irritants, as is familiar to every one who notes the effect of too continued poulticing. In distinction from cold, which is a nerve irritant, moist heat is a pure sedative. By a nervous irritant, we mean an agent which produces a lowering devitalizing impression, but which impression the nerves react against as soon as they can. By a pure nervous sedative, we mean an agent which produces a lowering impression from which the nerves may recover, but never react against; and precisely such an effect is caused by the contact of water at 85° to 95° to the surface nerves. You never saw a reaction against a simple warm poultice, nor does a patient come out of a warm bath with his heart more prone to vigorous contraction, than when he entered it; instead of that it may be enfeebled to faintness, and the most powerfully irritated muscles be so relaxed from paralysis of the irritation, that they will unresistingly allow a displaced bone to slide back easily into its socket. In diseases characterized by profound nervous exhaustion, such as tetanus, or hydrophobia, the slightest contact of water will frequently cause the worst possible centric disturbances. Now, my own experience leads me to rate this property of warm water as more certain and powerful than any agent with which I am acquainted for relaxing tonic contraction of either voluntary or involuntary muscular fibre, whenever such contraction is induced in response to sensor irritation. Repeatedly have I known it by itself to relieve and finally cure some of the most formidable instances of such muscular disorders. Four years ago I showed to our class at a clinic held in the Charity Hospital, Blackwell's Island, a woman who had both her lower limbs so drawn up that the heels had imbedded themselves in ulcerated depressions in her buttocks, and the adductors had so forced the knees together that the skin was wearing away from the condyles. Meantime the muscles of the legs were turned apparently into thin rigid whipcords. Every attempt to straighten the limbs caused her to cry out with pain; but, under ether, the limbs could be drawn down to their natural position, only to be seen slowly drawn up again, as the effect of the anæsthetic was passing off. She had not been able to stand on her feet for nearly two years, and yet I ventured to predict that, by a persevering use of the warm water douche alone, we would succeed in wholly overcoming her disability. The result proved as I expected; for, ten months afterwards, she walked out of the hospital herself, and returned to her former employment as a domestic. In the case of the laundress also, which I have just mentioned, the effect of this agent was equally gratifying. Even in those sad cases of tonic muscular contraction of the lower limbs, caused by pressure on the cord from vertebral disease or injury, the relief from this simple measure is often very great; in proof of which I would recommend you at your next visit to the Island, to examine a case of the kind in Male Ward No. 4, Charity Hospital. In our present case I expected the same results from the warm water, because the coldness of the limb and the characteristic painful rigidity with atrophy were all present; and I felt sure that if I could restore the arterial circulation in the left leg to the same degree with that in the right, I would not have much "sciatica" left to deal with; and the result, I think, has proved the correctness of this view of his case. I would, therefore, urge this procedure upon you for trial in all similar conditions, the douche being employed about half an hour to an hour after the anodyne injection, in order to start fairly with the adjuvant operation of the medicinal sedative. The object of the cold-water douche in this connection, is simply that of intensifying the nervous sedation by temporary excitation of the nerves with an irritant, much as a pedagogue deepens the impression of his chastisements by occasionally pausing for verbal admonitions. Cod-liver oil, the hypophosphites, nitrate of silver, and other nerve nutrients (in distinction from the proper quick-acting neurotics, which, as mere functional remedies never "cure" anything), are to be recommended invariably, on the ground that you cannot be certain how much organic damage may result, or have resulted, from prolonged functional nervous derangement.

What is the operation of the actual cautery in such cases? I can only answer that I do not know, and therefore cannot explain. The whole subject of counter-irritation is obscure, because its laws are linked to normal physiological laws of the nervous system not yet determined. The most universal remedial procedure among mankind is this of counter-irritation. I found it constituting more than two-thirds the therapeutics of the Bedouin Arabs; and Birmingham supplies the Chinese Empire with needles, not for sewing clothes, but for pricking patients. A Celestial colleague of ours will use from one to three papers of needles on a single hapless victim, with malarial fever. But how counter-irritation produces its remedial effects, we know as little as the Bedouins themselves. A much-esteemed member of our profession in this city was once incapacitated from all work for more than two years by sciatica. He had the benefit of the advice and services of a wide circle of his brethren, and he visited many healing waters in vain. At last he reluctantly consented to being burned for it; and he told me that, as he leaned over the back of a chair, and felt the hot iron passing down the track of pain, it felt not like what he had thought, but rather as the finger of a good angel. He got entirely well after some more of these angelic touches, and he could not be persuaded thereafter that there was any other cure for sciatica but this. Unfortunately I am not able to join in this comforting assurance; but you cannot tell until you have tried it how much or how little you are going to accomplish with it.

Case II.—Our second case is one which I have several times brought to your notice during the past winter, and which presents several instructive points of contrast to the preceding—Mrs. M. W., æt. 39, native of Scotland, a very intelligent person, and who has seen better days. Patient is a large, somewhat fleshy woman, who states that she has been from early childhood strong and healthy. As far back as she can remember, however, she has been troubled with constipation ; and when a school-girl, would frequently be a week or ten days without a passage of the bowels, while she usually went three or four days without an action, and then had to take some laxative. She was married when eighteen years of age, has had six children, but during her married life has had no special sickness nor womb trouble. Constipation, however, has continued, so that she has often been ten or twelve days without a movement, and then would require large doses of aperients, aided by enemata, before she could get relief.

She first commenced to complain of pain in her left hip, in the summer of 1870, when she would occasionally be seized with a sudden, sharp, spasmodic pain in the region of the sciatic foramen, while walking. These paroxysms would occur three or four times in a day and then disappear for a week, but she found that the pain would be instantly relieved by kicking or stubbing the toe against something hard. During this summer she did not feel very strong, and was rather below her usual standard of health. As the winter of 1870–'71 came on, the paroxysms of pain increased in severity and in frequency, and were not relieved by stubbing the toe as at first, while she had more or less pain all the time, and which was aggravated by walking. In the spring of '71 she commenced to "bloat," and was unable to go up stairs without inducing shortness of breath ; there was no swelling of the feet, however, and her account renders it probable that she suffered only from tympanitic distension of the bowels ; she had at this time, also, a troublesome sore-throat and tenderness on pressure in the bones. No reason whatever to suspect syphilis. During the winter of 1871–'72, the limb became very troublesome, so that she could walk but little, the pain remaining fixed about the sciatic foramen. In June, 1872, she went to Scotland, and while there improved very much, so that while in the Highlands she became free from pain altogether, and could walk as well as ever. A few days before returning to this country, however, the pain returned with great severity, after taking a long tramp and being exposed to the wet. Since her return here she has been under various modes of treatment, alternately trying regular physicians, homœopaths, electricians, and herb doctors. In the spring of 1873, an issue was put in the hip just above the sacral foramen, and she ascribes her increased sufferings since mainly to this application. She was obliged during its employment to keep her bed for five months, constantly lying on the right side, and ever since has not been able to get about without using crutches. The pain, however, had not yet radiated from its original seat to any other part of the leg, and during the whole period her bowels have continued greatly constipated.

Treatment commenced January 15th, 1875. Patient unable to walk more than a few steps at a time with the help of a crutch. Sitting is almost equally painful, so that she has to recline during the day as well as at night, and invariably on the affected side. If she lies for any prolonged period on the sound side, she has to turn over to the left and rest awhile before she can attempt to rise, and she has to be very deliberate in the acts, either of sitting down or of getting up. The pelvis is constantly raised on the left side. Face very expressive of pain, and her voice is pitched in a uniform complaining key. The pain is always referred by her to the sciatic foramen, and described as if a worm was steadily gnawing there; but, quite frequently, severe paroxysms of a sharp cutting kind are experienced, which occasionally, though seldom, radiate upwards, over the nates and down the leg. She has not noticed that wet or damp weather produces much change in her symptoms, but is quite certain that intense cold weather does, for she avers that she can tell, while still in bed, that the weather outside has turned cold, no matter how warm the room she is in may be, by the gnawing pain then acquiring a peculiarly deep character. On examining her leg, it is not found to be any different in size from its fellow, nor is there any atrophy about the nates. The temperature of the limb is also normal, although she complains that it constantly feels cold, and cannot be heated, even in warm weather. On testing with the esthesiometer, a patch of very marked anæsthesia was found, about four inches in diameter, corresponding closely to the distribution of the cutaneous branches of the lesser ischiatic nerve. Pressure at the sciatic foramen aggravated the pain constantly felt there, but did not cause any pains to pass downwards along the course of the great ischiatic, nor was there any tenderness in the popliteal space nor below it. The skin of the whole limb, however, showed diminished sensibility compared with the right.

The points to note here, are : First, a history of very chronic and obstinate constipation, commencing in early life, as she states, after an attack of scarlet fever, and continuing without change for years. She, however, has not suffered from hemorrhoids, nor from cystitis, nor from congestion or displacement of the womb, nor from disordered menstruation. She never had an attack after confinement, of perimetritis, which accident of parturition by occasioning intra-pelvic exudations or adhesions often gives rise to severe pains about the hip or along the great sciatic ; nor did she ever have a difficult or prolonged labor. I therefore early came to the conclusion, that the most probable exciting cause of the pain was the constipation itself. The relation of fœcal accumulations to the causation of sciatic pains, has been made out in so many cases, that you should carefully examine into the state of the bowels in every instance of this affection. You should not be satisfied also with the statement that the patient has a daily motion, but should ask what is its general character, and especially whether it is usually small in amount. Having done this, you should then make a careful exploration of the abdomen, not only to determine, as far as possible, the condition of the large intestine, but also whether there be any tumor like an ovarian cyst, or other abnormal feature present. Niemeyer quotes a case of severe sciatica, which seemed to be caused solely by a large collection of cherry-pits in the sigmoid flexure. It is difficult to assign the reason why, out of so many thousands of cases of fæcal accumulation from long-standing constipation, only a few ever have pains in the leg result therefrom ; simply because we can very rarely make out all the elements in the causation of nerve pains in any part of the body. But there are numerous physiological facts which go to show that the pelvic and visceral nerves generally in the abdomen, are intimately associated with the nerves of the lower extremities. If you severely pinch the folds of the mesentery in rabbits or in dogs, you will often note that it occasions complete paralysis in one or of both legs, and intra-pelvic irritations especially, are apt to produce reflex pains in the legs. A stone in the bladder sometimes gives rise to a pain felt in the sole of the foot. That, in this instance, the pain in the hip might at first have been owing to a reflected irritation, and, therefore, rather functional than organic in its nature, is rendered probable by the fact that then it could be promptly relieved by a jar to the foot.

The treatment ordered Jan. 15th was : A pint of Kissingen water before breakfast, and the iron mixture, which is such a favorite with me, viz., ℞ carb. ammoniæ ʒj ℈j ; ferri am. citrat. ʒj ; tr. nucis vomicæ ʒij ; tr. quassiæ, tr. gentianæ comp. āā ℥iv ; syr. simplicis vel elix. curacoa ℥iij : aq. camphoræ, ad Oj. Dose, one to two tablespoonfuls after each meal. The leg to be douched before going to bed with warm water for ten minutes, without the cold, and then rubbed well with olive oil, with a few drops of the oleate of mercury applied to the seat of pain. Jan. 19th. Patient complains more than before. Says that the rubbing of the leg aggravates the pain. Weather intensely cold. Ordered an injection of six drops of Magendie's solution, and $\frac{1}{60}$ grain of atropia every night. Omit the douche and rubbing with oil, but continue the mercurial oleate. Jan. 29th. Weather 8° below zero last night, and patient suffered severely. Unable to sleep; says that the whole body feels sore; has some fever and headache, but the headache was relieved in a few minutes after the injection of gtt. xv. Magendie's solution with atropia as before. Feb. 6th. Treatment so far has afforded no relief, and the patient is worse. The Kissingen has wholly failed to act on the bowels. Ordered to omit the inunction of the oleate; to substitute a prescription of quinine and sulphuric acid for the iron mixture, and to begin a course of purgation as follows: one drop of ol. tiglii in a drachm of ol. amygdal. dulc. every night, while the hypodermic injections continue at night as above. Feb. 13th. General condition very much improved. Has three or four watery passages a day from the croton oil. Is decidedly relieved from the pain about the hip, and is able now, for the first time in nearly two years, to get up directly from a chair or sofa without being compelled "to turn in getting up." She can also walk without crutches, sleeps well, and can turn herself in bed better than at any time since the issue was used in the spring of 1873. Feb. 19th. General condition and relief from pain about the same as at last report; but she begins to feel somewhat prostrated from the continued purgation by the croton oil. Feb. 26th. Is beginning to suffer considerably from the continued purgation. The croton oil acts now very quickly, within an hour or two after taking it, when at first it did not operate till morning; and as it calls her up in the night, her sleep is very incomplete. She, therefore, has become nervous and easily excited; and, although the hip pain is much better, yet she is less able to bear it. Ordered to take a mixture of valerianate with muriate of ammonia during the day when depressed, and to take the croton oil in the morning instead of at night, and to have five touches with the hot glass rod about the sciatic foramen. March 7th. Patient has been much more comfortable since last report. Ordered to-day to omit the morphia and atropia injection, and to commence with an injection of coneine, one-sixth gr. and ergotine grs. iij. in twenty minims of water directly at the seat of pain. The first injection was followed by much local pain, and on commencing another cauterization a few minutes subsequently, like the preceding, she was seized suddenly with a severe "griping" pain about the heart, accompanied by a good deal of fluttering, and which cardiac pain lasted more or less till morning. The injections of ergotine and coneine were continued daily for five days, but the cardiac pain recurred each time, and was not relieved by Hoffman's anodyne and camphor given several times a day. Each injection also occasioned a local inflammation, which made the whole limb so painful that she ceased to walk again. In four days after the omission of these injections she was greatly improved, and walked down a pair of stairs without difficulty for the first time in six months. She has now no spasmodic pains, only the old steady gnawing pain at the original site, but much less of that. March 27th. Ordered the cauterization again on the 22d, and to omit the croton oil, as she felt so weak from its action. But, a few minutes after being cauterized, she was seized with a sharp lancinating pain which ran from the hip to the ankle; the whole leg became very tender and continued so, with paroxysms of the radiated pain, for three days; the patient becoming much discouraged thereat, as it was a new experience to her. Ordered to take a suppository of ext. stramonium, gr. j; ext. belladonnæ, gr. ½; morphiæ sulph., gr. ½, each night; and to recur to the mist. ferri. March 31st. A bath of sulphuret of potassium, ℥xij; salt, one pound; to thirty gallons of water was now ordered to be taken every night. April 14th. Took the baths for eight nights, and seemed much relieved by the first four of them, but the skin then began to become irritated, and sores formed of a superficial kind, feeling very much like burns, on the top of the foot, on the shin-bone, on the knee, and one large one corresponding to the anæsthetic patch over the sciatic notch above referred to. The skin of the whole leg became very tender and sensitive; and on a cold day (April 13th) she had severe darting pains down the leg, and especially at the ankle, which "was like a toothache." At this time the skin of the other leg also became very sensitive to the touch, and she complained that she could not keep them warm. Has resumed her crutches. April 24th. Patient resumed the purgation with croton oil at the last date, to be continued till her stomach would become uneasy, when she should omit it for a few days, and then resume again. She has decidedly improved again, and feels better than at any time during treatment.

Now, in this case, I started with the theory that a localized perineuritis existed just within the sciatic notch, which occasioned some pressure on the lesser ischiatic nerve, as it emerges below the pyriformis muscle, and this view was based upon the pain being felt most on standing and attempting to turn the body on the hip, or in raising the leg to the top of a chair and making an effort to step upwards, in which act the pyriformis and the gemellus muscles must swell in contracting so as to press upon the sciatic nerves in the notch. This supposed perineuritis was ascribed to the state of the pelvic circulation induced by the long-standing constipation, aided by the weakening of the portal circulation common to the later periods of adult life, and frequently aggravated in women by the approach of the menopause. The oleate of mercury was applied for the purpose of assisting in the absorption of effusion or plastic exudation, for I have considerable confidence in its effectiveness in such conditions, notably in rheumatic stiffness, either articular or muscular. The warm water douche was intended solely to soothe pain; not, as in the former case, to relax arteries—for there was no indication of this kind present here—and hence the application was to be once and for ten minutes only in the day. The Kissingen was to remove portal congestion, and a thorough course of such a saline mineral water has about as great and well-deserved a reputation for curing sciatica as any other remedial measure whatever. But the patient grew worse. The reasons for this, I think, are plain; namely, that the mineral water failed *in toto* to have any effect; and hence the most important indication in the case, because it was directed to the primary source of the mischief, remained unfulfilled. In the next place, the warm water, instead of relieving pain, as it does in the great majority of instances, seemed rather to increase the patient's peculiar susceptibility to cold weather. This susceptibility was not properly of the rheumatic kind, for the invariable characteristics of that are increased pain on the approach of a storm or similar atmospheric disturbance. It was, however, only intense wintry cold, and not snow, rain, or damp which this patient felt; and experience will teach you that certain nervous derangements are much influenced by cold alone. During very cold weather the obituary columns of the journals will show a decided increase in sudden deaths from apoplexy, and the majority of these cases will have occurred, not while exposed to cold directly, so as to be explained by internal congestions from the surface being chilled, but after the individuals have been lying for some hours in bed.

Another feature of this case, and a somewhat unexpected one, was the evil effects of counter-irritation in any form, designed or undesigned.

It is especially in cases where you suspect pain to be dependent upon inflammatory processes, more or less, that counter-irritation proves most often serviceable; but, for reasons soon to be noticed, I did not pay much heed to the patient's certainty that the issue employed before had made her a great deal worse, and so tried the hot glass rod four times, with the result of awakening a nervous tempest on the two last applications. Moreover, whenever soreness was occasioned by the hypodermic injections, she complained that her spasmodic pains were increased, and notably so on the occurrence of the cutaneous irritations which were caused by the sulphur bath. In examples of rheumatic sciatica, sulphur and salt baths often relieve the patient greatly, and though I did not suppose any rheumatic element to be present in this case, nor do I believe that sulphur can have any other effect in a bath than by its impression on the surface-nerves, for not a particle of it can be absorbed from a water bath, yet I thought the probability of some good resulting from it justified its being tried. The result, however, satisfied me that *all* attempts to relieve this case by peripheral impressions were wholly useless. The mere rubbing with olive oil seemed to increase the irritability, as if the surface-nerves were in such an abnormal state that they were not capable of influencing the nerve centres in any of their usual ways ; and hence, if relief were to come at all, it must be by acting on the proximal, rather than on the distal division of the nerve made by the affected locality.

In striking contrast, however, with the ineffectiveness of all these measures in this case, were the results obtained from the active purgation by croton oil. In fact, this was the only real remedy here ; and so often have I succeeded in relieving sciatica by purgation, and notably by the powerful operation of this cathartic, that I speak of it now, as, on the whole, the most generally applicable resource that we possess in obstinate cases, and that, of course, means in most cases of sciatica, for it is a complaint which the Adversary might well have tried on Job, if his design was to wear out human patience. In Ringer's "Therapeutics," under the head of "Croton Oil," you will find a reference to the recommendation of this purgative in sciatica, by Dr. Sewell, of Ottawa, Canada. Dr. Sewell lays great stress on the evacuation of blackened fæces by croton oil ; and he says that its operation will cure patients with this complaint who have not been constipated. I have not found the discolored discharges Dr. Sewell speaks of in my patients yet, but I can bear testimony that it does relieve those who have not been specially constipated, as well as those who have ; but they have always been patients who showed some sign or other of defective abdominal circulation.

The last point I would allude to is, that our patient's case was one, which for brevity's sake, I would term a female nervous system, about forty years of age. You will soon find out that there is such a thing as a female nervous system, and female nervous diseases, not necessarily connected with the female organs of generation, but rather with those wider differences from the male sex which obtain in the physical and nervous organization throughout. In sciatica you will meet with examples among women in which it may be hard to say whether, if they had the nervous system of a man, they would have had the disease at all. I do not mean that these should be termed cases of hysterical sciatica, for, as a rule, they do not present other more distinctive manifestations of hysteria, but they will, nevertheless, trouble you with accounts of their various and perhaps inconsistent symptoms, in which the emotional element will predominate to such an extent as to lead you to distrust them, very much as you distrust the aches and pains of hysteria itself. This patient whined constantly. She felt pain more than a man would, and it was easy to suppose that she had those female sufferings in abundance which we meet with so often, and which seem to be as much subjective as objective. On that account I did not pay much heed to her story about the issue having made her so much worse, but afterwards I had reason to feel that her views were more correct, in this particular, than mine. Do not, therefore, let that will-o'-the-wisp, hysteria, lead you astray too often ; only keep your judgment in suspense until your well-considered measures of treatment, by their results, help you in your diagnosis. We have no time now to indicate the bearing which the great revolution of the menopause has upon the evolution of nervous derangements ; but I will simply say that derivation by the bowels will answer oftener than any one other general measure in the treatment of the protean ailments of women at that period, neuralgic affections included. Always follow this up, however, by concomitant measures for blood renewal, such as by iron, phosphorus, and notably, cod-liver oil or cream, and be very guarded in your recourse to the deceptive aid of mere neurotics,—whether they be special stimulants like ether, ammonia, valerian, and assafœtida, or, on the other hand, the anodynes. You might as well treat chronic starvation by stimulants or narcotics, as chronic neuralgias by neurotics. I wish you to notice, lastly, that five months' rest in bed did not help this patient ; nor, unless her bowels were acted upon, do I think she would have been cured by resting the remainder of her life. Rest is a great adjuvant in treatment of sciatica, but an adjuvant only.

We have no time, owing to the length of our remarks, to do more than allude briefly to some points in the history and treatment of the remaining two cases, which we now bring before you. The first is that of F. R., aged 27, American, truckman. Patient's general health good, but admits having had both gonorrhœa and syphilis, and that he occasionally gets drunk. About six months before his last admission to this hospital, while at heavy work in lifting and rolling boxes of bacon, of 500 lbs. each, he was suddenly seized with a severe pain, shooting down the back of the left leg, from the sciatic notch to the middle of the calf. The pain was so great, that he says, "he could have bitten a nail in two, if it had been between his teeth." For the next two months he suffered all sorts of agony, and was entirely prevented from work. He had no numbness nor loss of sensation, but had repeated painful spasms of the muscles of the calf. Two months after the attack he was admitted here, in the Third Medical Division, under the charge of my colleague, Dr. Austin Flint, and remained one month under treatment by blisters, and subcutaneous injections of morphia, after which he was discharged apparently cured. Four weeks afterwards pain recurred upon some unusual exertion, and he was admitted again in the Fourth Medical Division, under my colleague, Dr. Gouverneur Smith. He now received in three weeks three separate subcutaneous injections of ♏xx of chloroform. After the first injection the pain was relieved for two days, and then returned, but of a dull, aching character, instead of the sharp, shooting kind, which had troubled him before, and the same experience was repeated, after the other two injections. On Feb. 19th, he was transferred, at my request, to the Second Medical Division, under my care. Feb. 22d. Patient complains of a large elongated swelling just internal to the course of the sciatic nerve, which is not hot, but rather hard and elastic to the feel, painful on pressure, and accompanied with a sense of numbness in its neighborhood. Ordered to have the swelling poulticed, to have subcutaneous injections of $\frac{1}{60}$ gr. atropia, and ♏viii Magend. sol., and the hot douche to the whole leg for twenty minutes, three times a day, and to rub in the oleate of mercury at night. Feb. 25th. Much improved, swelling and pain less. Ordered to continue above treatment, and in addition, to have the leg well oiled after the douches, and to take a pill containing one drop ol. tiglii, and $\frac{1}{8}$ gr. podophyllin every night. Mar. 11th. Patient steadily improved under this treatment, though the pills had to be omitted occasionally on account of nausea. He had two relapses which he ascribed to not having hot water enough for his douches, as he experienced most relief from this measure. At this date he seems to have felt so well, that he went out of the hospital without leave.

You will note, that in distinction from the usual history of sciatica, which, as a rule, comes on gradually, this man was attacked first very suddenly, while the muscles of his leg were probably strained to the utmost. Straining during defecation is also enumerated as an exciting cause of sciatica. A case of this kind, therefore, is virtually like a traumatic injury of the nerve not dissimilar in character to that of another truckman who recently came to my college clinic with the history of pain, beginning from the date of a sudden jolt on the high seat usually provided on trucks for the drivers. If you are called in soon after the injury, I would, therefore, recommend leeching about the sciatic foramen, or to the anus, to begin with, then injections of morphia and atropia to relieve the pain, and lastly the oleate of mercury, enjoining also perfect rest for several weeks. Effusion into the sheath of the nerve is doubtless a common accompaniment of these accidents, and the hot water douche, beside relieving pain, increases absorption owing to its effects on the local arterial circulation, while the same may be predicated of the action on the bowels.

The other case is that of Owen McG., aged 60, Irish, dock-builder. Patient states that his general health has been excellent, denies venereal, but admits that he drinks habitually, though not a drunkard. Last October, four months prior to admission, he had to stand daily for prolonged periods in water while at work on a dock. He then began to notice a pain of a gnawing, aching character, commencing at the sciatic foramen, and slowly passing down the back of his left thigh and leg to the ankle. This pain is never violent, and he does not feel it at all while sitting still, but experiences it immediately on moving. The leg feels to him as if it were asleep, and in trying to use it it seems much heavier than its fellow. He complains that he cannot keep it warm, while the sensation of hot water is appreciated a great deal sooner in the right leg than in the left. On testing for painful points along the divisions of the sciatic, it was found that he had very little pain at the sciatic notch, but severe pain was caused by pressure on the sural nerve which extended to the external malleolus and foot, while another region of pain was on the sacrum, and extended to the crista ilii. He always grew worse in damp or stormy weather. He remained under treatment from Feb. 22d till April 15th, improving and then relapsing according to the weather. He obtained the most permanent relief from hot water douches, and latterly from sulphur and salt baths, used as in Case II., with occasional cauterization of the painful localities. He was peculiarly susceptible to the injections of morphia and atropia, which made him dizzy, and blurred his vision much more than they afforded relief to his pain. The sulphur and salt baths, however, appeared to benefit him from the first trial, and as he thinks, bid fair to cure him.

This case, both in its causation and symptoms, is a typical example of what may be termed rheumatic sciatica. My experience does not lead me to regard the rheumatic form of this complaint as by any means so much more common than the others, as you might infer from the text-books. I must say also that the medicinal treatment usually recommended for rheumatic disorders has not proved very efficacious in my hands in rheumatic sciatica. A few cases seem to be helped by iodide of potassium with fluid extract of conium, but with many it fails altogether. Local treatment on the other hand by faradization is frequently beneficial ; but on the whole I would recommend a persevering trial of the hot douches, and sulphur baths, counter irritation and the relief of pain by anodynes.

This leads me, in conclusion, to say a word about the place of hypodermic injections of neurotics in the treatment of this complaint. I rarely expect to cure sciatica by any of them, for the simple reason that in the great majority of cases it is a very chronic and obstinate affection, and therefore you should not expect to master it by the employment of mere functional rapidly-acting and rapidly-ceasing remedies like the neurotics. All such agents deal with the symptoms only, but not with the causes of the symptoms. Morphia, atropia, chloroform, conia, ergotine, etc., may each prove of great service as adjuvants, but if you think that you can permanently remove any old standing neuralgia by them alone, you will generally find that you simply diminish the pain felt, because the aching nerve cannot act like a living nerve for the time being.

Ere long also the nerve becomes able to ache in spite of the hindrance of the first dose, and you may be obliged to enter upon a most unsatisfactory course of educating it to bear the anodyne indefinitely. You have often had your attention called to the case in Ward 25, of the man who, for neuralgia of the superior maxillary nerve, had taken hypodermics of morphia so constantly that after he had been long free from pain under treatment by arsenic and the glass rod, yet he would beg for the morphia injection, like an old opium-drinker. You should therefore use neurotics in this disease just as you may use ether, camphor, alcohol, or opium occasionally in the course of typhoid fever, not to cure the fever by them, but simply to relieve symptoms or to prevent complications

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OTITIS; OR INFLAMMATION OF THE EAR,

AND ITS RELATIONS TO WHAT IS COMMONLY CALLED "TAKING COLD."

BY

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At our last meeting we were upon the subject of otitis media catarrhalis, catarrhal inflammation of the middle ear; to-day, I propose to follow the line started at that time, and in the simplest and most colloquial manner to point out how the general practitioner may, by obeying a few plain rules, treat the cases of acute disease of the external and middle ear that are certain to occur in his practice. I shall not speak as to aurists or experts, but address myself to you as medical students and general practitioners. I shall try to avoid such allusions as would be understood only by those who are familiar with aural medicine. My purpose is to help the general practitioner to prevent some of the evil results which now so frequently come from the tardy, timid, or ignorant management of such aural affections as spring from common catarrh and furunculosis, or arise as complications of sore-throat, measles, or scarlet fever.

We have before us now a man in middle life, a teacher, who lost his hearing by exposure to cold. He, no doubt, had, at first, an otitis media catarrhalis, catarrhal inflammation of the middle ear. We need not repeat the history of the case. It will serve our present purpose to accept that diagnosis as the basis of our remarks. It is the so-called taking cold which gives us many of the cases of acute or recurring middle ear disease. If this is so, it becomes an urgent question how may this tendency to catch cold be lessened or prevented. There is an additional pertinency in the inquiry to-day coming from the fact that the thermometer is ten or twelve degrees below zero, and catarrhal affections of the throat, ears, and chest are very prevalent. When we catch cold, or get a more or less acute catarrh, it is generally traceable to a sudden or unexpected fall in the temperature of the air, or to exposure of a limited portion of the body to moisture, or to the cooling effect of air in motion, or to the depressing influences of overheated or impure air in a dormitory or public assembly room. As we often cannot have the best conditions of ventilation and heating, how may we prepare the body, not only to withstand the inevitable exposures, but to turn even a draught into a pleasurable source of health. Living, as we ordinarily do, without sufficient out-of-door active exercise, the surface of our body becomes morbidly sensitive and susceptible to all those influences which produce catarrh. Hence you shall find among people the greatest dread of a draught. The average man, in a public vehicle or assembly, would rather incur the risk of typhoid fever than endure a draught. And there always is one or more enemies of his race at every discovered inlet of fresh air in an assembly room, ready, even to break up a public meeting in an uproar if the influx of the needed air is not regulated by his individual sensibility. Air in motion is evidently regarded by too many as one of the most morbific influences that a man at rest can meet. Hence the great obstacle in the way of ventilating public vehicles and buildings. Very few persons have been so educated as to endure, when at rest, air in motion, much less to derive pleasure and health from it. Now, I know from personal experience, and many years of close observation and practice, that it is quite easy to correct or cure this morbid and often fatal sensitiveness, this tendency to fall an easy victim to the causes of common catarrh. If I am right, and the simple rules which I shall give produce the results in your practice that they have in mine, we shall hasten the time when a sufficient number of persons will be found in every community able to endure draughts, to insist upon the admission of sunlight and fresh air wherever human beings are domiciled.

Now, how may you and I learn to endure draughts and lessen our tendency to catch cold? First, by diminishing the morbid sensibility of the surface of the body. This can be brought about by graduated exposure and friction of the skin in a daily air or sun bath, followed by such local sponge baths as you may be able to speedily react from. That this reaction may be quick and spontaneous the temperature of the water used must not at first be much lower than 80° Fahr. It is well also at first in the air bath, to expose the body for a very short time only, such as would be spent in walking briskly across an ordinary bed-chamber. After a little practice the length of the exposure may be increased to fifteen or twenty minutes and the temperature of the water used in washing accommodated to that of the outer air. The salutary effect of this exposure may be still farther increased by two or three deep, chest-filling inspirations, with closed mouth, and by a few such movements of the arms as would tend to invigorate the chest muscles and quicken somewhat the action of the heart. At least four times a week, while taking the air bath and *before* any water is applied, the entire surface of the body should be rubbed briskly with "hair mittens and strap" until there shall have been produced a sense of glow and warmth of the skin. If the subject be not too feeble, the rubbing should be done by himself rather than by another, as it is thus made more beneficial. At first these exercises should be very brief, especially if the heat-producing powers of the body are low. In the latter case they should not be attempted at first, especially in the winter, except in a sunny room or in one artificially warmed. Under all circumstances it would be better to use a sunny room. It should always be kept in view that the object of the treatment is to gradually and systematically lessen the morbid sensibility of the body, by *daily* exposing the *entire* skin surface to air, light, friction, and cleansing, in an atmosphere as nearly as possible at the prevailing temperature. Of course, imprudence in exposing the untempered nervous system of the skin for too long a time to a low temperature would defeat the grand purpose of the training and bring the method into contempt. Invalids should not plunge into the full practice at once, but enter upon it deliberately and cautiously, as one ignorant of swimming would wade into the water. Observing the cautions given, I believe that almost any one may, in a few weeks, render himself not only less susceptible of taking cold, but also better in every way, the benefit coming from the extensive range of sympathetic relationships which exist between the skin and every other part of the body. I do not say that persons thus trained may, for instance, run to catch a ferry-boat and then, without overcoat or hat on, stand with impunity exposed to a north-easter. They will be as cautious in times of danger as need be, but a love of pure air in motion will so take the place of the former dread of the same, that insensibly they will find themselves armed against the many foes of health that infest the air of domiciles and lurk in atmospheric changes. This part of the preventive treatment of catarrhal affections of the ear would be very incomplete, if we did not touch upon three or four other hygienic points that have important relations in the causation of "taking cold." I refer to food, clothing, and exercise. I am thoroughly convinced that the re-action from even moderate doses of alcoholic stimulants increases the tendency to "catch cold," and that one who has taken an alcoholic drink under the delusion that by so doing he may keep the cold out, should instantly put on an additional overcoat to keep his animal heat in. This conclusion is entirely irrespective of the question as to whether alcohol is food or not. If it be food, I believe it is, as a rule, very bad food, except in certain states of the body—and what those states are, even the most learned chemists and physiologists have not yet determined with anything approaching agreement.

Experience in Arctic, and other naval and land expeditions, involving severe wear and tear of the body and exposure to climatic effects, whether of heat or cold, go to show uniformly that those who use alcohol suffer most from disease. The testimony of the English Parliamentary reports is remarkably unanimous on this point, a testimony that is drawn from the widest colonial experience, as England has had her flag under every climate. And here let me say in passing, that the best restorative for one who has been chilled by exposure to cold is hot water with or without milk, or black tea. Let one who wishes then to do everything possible to lessen his tendency to catch cold, avoid alcohol—even in small doses. Every article of food that merely excites the nervous system without helping materially to make good tissue, belongs to the same category. Every article of food that induces dyspepsia should be avoided, such as greasy potato-hashes, "fries," and many of those sapid messes that garnish the tea or supper table. The stomach and digestive organs should be trained into vigorous action. A man who cannot teach his stomach to be a good and provident servant of his body, is to be pitied indeed. A man with an uneducated, sour, or flatulent stomach is in a bad way generally, and especially whenever, as in the experience of the memorable and susceptible Mr. Jarndyce, the wind is out of the east.

I think that every one should try to learn to eat coarse farinaceous food and milk. Such articles of food as crushed wheat, wheaten grits, oatmeal, beans, and peas, and milk should enter freely into the dietary. Many of these articles of food not only contain indispensable tissue-building ingredients, but by their mechanical contact with the digestive organs do for them a work something like that which friction of the skin with the "hair mittens" does for the surface of the body in hastening the desquamation of effete and sticky epithelium and the cleansing of follicles. I would recommend then a simple breakfast of thoroughly boiled coarse farinaceous food, with cream or milk, fruit, and eggs. A lunch of somewhat similar character, and a hearty dinner, free from condiments and spices, but generously made up, if possible, of several courses, including fish, meat, and vegetables. Spices should be used very sparingly, if at all, especially by the young, and rather reserved in diet, to wake up the powers of senile mucous membranes. There is a form of chronic and intractable inflammation of the fauces, that frequently leads to ear trouble, which seems to me to be aggravated, if not induced, by the use of spices and tobacco, and by taking even otherwise good food, at too high a temperature. We should learn to eat food more slowly, to masticate it better, and to take it at a temperature more nearly that of the body. We come now to speak a little on the subject of clothing. Woolen flannel graduated in thickness, according to the season, should be worn next to the skin, by day and by night, throughout the entire year, in every climate. This rule applies to all ages. The prevalent idea that children can be "toughened" by insufficient clothing, and a promiscuous dietary, is a destructive fallacy. Even otherwise intelligent persons draw reasons for the practice of keeping the legs and chests of their children bare, and going themselves without flannel, from the supposed good health of savages and paupers. The fact being, that in both of the classes so absurdly quoted, the mortality among children and adults, which is excessive, is largely due to exposure as well as unsuitable food. This underclothing should always be changed at night, and never worn for more than two consecutive days without being washed. Better have a wardrobe rich in abundance of underclothing, than in so much mere outside decorative dress. If the same underclothing be worn day and night, the skin will absorb impurities which had previously been excreted. Some may smile at this caution, as though nobody ever wore the same underclothing day and night. Make more careful inquiry about the habits of your patients and you will know better. If you are compelled to wear any of the same clothing on the following day, turn such pieces, whether under or outside garments, wrong side out, and hang them separately, that they may be thoroughly dried and aired. Clothing so treated will last longer, an economic fact that begins to be not below the consideration of even an "American citizen," and will be much freer from animal impurities. The neck-ties and bands should be so loose as not to obstruct the return circulation in the neck ; and foot-covering should be thick, loose, and low-heeled. As one of the means of guarding against catching cold, the foot-covering should be so broad in the soles as to leave the interosseous spaces in the feet free to protect, without pressing upon the nerves and vessels that vivify the toes. Many a cold, especially among women, is "caught" through feet that have been compressed into a partially lifeless mass by a narrow-toed and high-heeled shoe. The sole of a shoe should be as broad as the foot, and the height of the heel should never be, even for a thick sole, more than five to six-eighths of an inch high, measured behind. High-heeled shoes not only injure the feet, but, by throwing the base line of the body in front of the arch of the foot, bring the arms, by gravity, too far forward, and, tilting the whole frame in the same direction embarrass the chest, preclude free inspiration, bring an unnatural strain to bear upon ligaments, muscles, and other important organs, and thus make out-of-door life irritating and speedily fatiguing. Thus women and others who should be merry pedestrians, and ready even to contend with stormy weather, ride about, the flabby occupants of padded carriages, the victims of neuralgia, and other endless disorders that tend to fatty degeneration, catarrh, pneumonia, and premature decay.

A few words more regarding exercise as a means of guarding against "colds." The best of all forms of exercise is walking. Any one possessed of legs can learn to walk, and yet you shall meet patients every day who tell you that they cannot walk. Walking for pleasure and bodily profit has, with too many, become a lost art. You may teach any one who is not the subject for an immediate surgical operation to walk. The proper method is to set tasks of walking. Prescribe a daily walk, so short at first as to be clearly within the limits of the physical powers of your patient. Say one city block or a thousand feet out and back, and order a specified number of feet to be added daily to the length of the excursion. In this way I have induced very many to develop their locomotive powers and to gradually grow strong enough to walk miles with the greatest possible benefit to health. To secure such results you must be specific in your directions, even to the point of writing them down—many will say that they cannot walk without an object. The mere pleasure of locomotion should be a sufficient object, and would be, no doubt, if the organs of locomotion had not been crippled by disease or bad covering. If natural history and drawing were more generally taught, there would be an insatiable fondness for out-of-door life. People would have their perceptive faculties so quickened that they would observe what they saw and find more real pleasure in searching for the objects of nature. Many a college graduate in our country knows little or nothing about nature, as its works are spread everywhere about, and gets little or no pleasure or instruction from out-of-door life. If this be so, how must it be with those who have had very few opportunities for culture. One soon gets tired of merely looking over the sea or landscape unless those objects have been so studied as to be filled with inducements to pleasurable thinking, and thus to gratify some of the higher cravings of our nature. To sit in aimless indolence on the piazza of a watering-place hotel, or drive in state on a frequented or dusty road, gazing at the inmates of passing vehicles, is certainly not likely to produce a fixed and ruling determination to spend daily, some portion of time in active out-of-door exercise.

We have thus far tried to touch upon some of those hygienic matters which are related to one of the commonest causes of ear disease, namely, catarrh, either in its acute or chronic form. We have said very little about medicines. Our object has been to show how observance of the rules of hygiene would tend to lessen the tendency "to catch cold." We have tried to address ourselves to you as students and to be as simple and even colloquial as possible in enforcing what we believe to be correct hygienic principles.

We pass now from that which is preventive, to discuss some of the conditions of disease which demand promptness on the part of the practitioner in his efforts to cure external, or middle ear inflammation at the earliest moment. We will not go over the various diseases systematically, our purpose not being to make a treatise for aurists, but to interpret for the general practitioner some of the more urgent and significant symptoms of acute external or middle ear disease, and to show how the indications may be so obeyed as to lessen the number of instances in which acute disease, unrecognized and unchecked, ravages the organ of hearing. First, then, we would say that every medical man should be able to distinguish the drum-head or membrana tympani—not one in a hundred can. Five minutes, spent daily for a week, in looking into the external auditory canal of a healthy ear, through a conical speculum, would enable one with ordinary powers of observation to avoid many of the catastrophies in treating common ear diseases.

"Ear-ache," so generally maltreated, comes, commonly in the beginning from one of two forms of ear disease, either inflammation of the dermoid or periostial lining of the external auditory canal, or acute inflammation of the middle ear. In these affections one may safely meet the urgent symptoms without making a very precise diagnosis, and thus save many ears.

Pain and deafness often occur in the course of common boils in the external auditory canal. How may we then easily distinguish such a disease from something deeper? Every one of you should be able to tell, at sight, whether a given external auditory canal is changed in calibre or not. If changed, what is the nature of the change. After inspecting the canal, explore with a probe, guarded with a pledget of cotton, to ascertain whether there is a focus of local inflammation. Carry the end of the probe around the entire circumference of the canal, touching each segment of the same. The instant that the end of your probe shall have touched the inflammatory centre, or boil, your patient will give audible evidence that you have found the tender spot. Repeat this experiment once or twice, and then, with a sharp, curved bistoury or knife, like that in fig. 2, freely incise the inflammatory centre down to the bone. Do this early, before the boil has involved much of the surrounding tissue, and you will stop, in many instances, that evil communication which might otherwise cause a succession of boils. After the incision, foment with a stream of hot water, let into the external ear from a fountain syringe. Never apply poultices under these circumstances, as the uninterrupted application of heat and moisture tends to beget œdema, making more or less of the lining of the canal boggy, and favoring the production of new boils, or of an obstinate, diffused inflammation. If this early incision does not cure, apply leeches, and follow those with hot or warm fomentation. The best place at which to leech for affections of the external ear, and ordinarily for those of the middle ear, is in the hollow at the base of the tragus, half an inch in on the front wall of the external auditory canal. Apply the leeches with a glass tube, having previously passed a plug of cotton beyond and scratched the objective point in the skin to start a little blood. One or two leeches so applied will do more good than when applied in front of the tragus, or over the mastoid, unless there be a mastoid cell inflammation that is beginning to outcrop behind the external ear. Even then it is well to divide the number of leeches to be applied, so that several may be put on over the mastoid, and one or two in the hollow inside of the tragus. Promptly incise every furuncle that occurs.

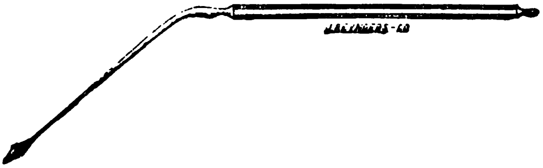
We pass now to speak of certain occurrences in middle ear inflammation requiring prompt attention. A man is taken with more or less deafness and pain in his ear, having been exposed to cold, had a nasal catarrh or sore throat, or been using a nasal douche. Perhaps he has had a night of pain or agony, and calls you early in the morning. What are you to do? Order a poultice over the painful organ, and an anodyne internally, and tell your patient that he must wait until "it bursts;" or, if the patient be a child, tell the attendant "that the poultices are to be repeated until there is a discharge?" If you do, I hope that you may get your discharge, and your place be filled with a more intelligent practitioner.

Cases maltreated in that manner form the bulk of the incurable cases of otorrhœa that fall into the hands of the scientific aurist, to worry him by their obstinate character, or to end in fatal temporal bone disease.

Called to such a case as we have alluded to, examine carefully the external auditory canal, to see that there be no inflammatory centre there, external to the drum-head.

Take a measure of the hearing distance with your watch. Apply leeches at once in the hollow, inside the tragus. Favor bleeding from the leech bites for an hour or two by hot fomentations. If marked relief of pain and deafness does not follow within a very few hours—say three or four—incise the drum-head with a fine straight bistoury, or a knife, such as we have in fig. 1.

FIG. 1.



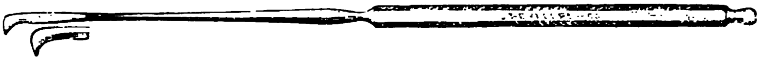
It seems to me that any one who can perform the operation of introducing a catheter, or pass a key into a night-latch, can incise a drum-head. It is only necessary to introduce as large a conical speculum as possible into the diseased ear, and then to pass the cutting instrument along the floor of the meatus, until the lower portion of the drum-head is reached and perforated. If possible, carry the incision from just below the extremity of the long process of the malleus to the lower margin of the membrane. After the incision inflate the ear, if possible, by Politzer's inflator, or, if that is not at hand, by asking the patient to hold his nostrils closed firmly by means of his fingers, and then to blow strongly, with closed mouth, into the nostrils. We do not agree with Gruber as regards the impropriety of syringing after incising the membrana tympani, if his objection is aimed at such cases. On the contrary we like to syringe the external auditory canal with a warm aqueous solution of common salt, or carbonate of soda, say a drachm of either to a pint of water. By so doing you will often hasten the discharge through the incision in the membrana tympani of the inflammatory products in the middle ear. Sometimes these latter are serous, more or less viscid and stringy ; sometimes sero-sanguinolent or even purulent. After this incision give anodynes freely — *not before ;* they may, if given before, mask the processes going on in the ear, stupefying your patient until "something bursts," that "something" generally meaning, to the intelligent, a more or less helpless rent in the membrana tympani ; or, if that does not occur, a thorough invasion of the mastoid cells. Stand ready now to repeat the leeches, and the paracentesis as often as may be necessary to throttle the inflammation. Do not be afraid of injuring the membrana tympani, even though you should repeat the paracentesis every day or every other day for a week or more. You cannot, by repeatedly incising the membrane, do it one tithe of the harm that may be done by leaving inflammatory products dammed up in the drum cavity until they macerate its machinery, threaten the portals of the internal ear, stuff the mastoid cells, or break through a more or less disorganized drum-head. *Be alert in these cases and strike your best blows at the inflammation within the first twenty-four hours.* Your weapons then are as follows, and to be usually used in the order named : leeches, warm fomentations, paracentesis, Politzer's inflator, anodynes. And, when you use the latter at the right time, use them boldly in doses to subdue pain.

Generally, persons suffering from " ear-ache " need not be kept in the house except for a few hours, unless in very bad weather. Moderate slow walking lessens the pain and seems, whether from the posture of the body or the influence of that form of locomotion upon the circulation in the head, to quicken the healthy process of resolution. If the pain is very severe, give the anodynes and encourage the sufferer to walk about slowly until a decided sleepiness has been induced.

The grand object in this stage of the disease is to maintain a free vent or outlet for the inflammatory exudations of the middle ear through an artificial opening in the drum-head. To be sure of this you must see your patient every five or six hours during the first two or three days and repeat the paracentesis of the drum-head whenever you have assured yourself that the opening previously made has closed. A good method to obtain this information is as follows. At each visit, gently syringe the external auditory canal with a little warm water holding some common salt or carbonate of soda in solution, say a drachm to the pint. Then inflate with Politzer's inflator; if you do not get a " perforation whistle," that is, the sound made by the air passing up through the Eustachian tube, through the middle ear, and out of the perforation, repeat the paracentesis. If possible, pass your knife through the closed or clogged opening previously made, if not, then through the lower part of the drum, below the malleus. Occasionally, the inflammatory contents of the drum cavity will be too viscid or stringy to escape, even when Politzer's inflator is used. The more prolonged expiratory effort made by the patient's blowing his breath strongly into his closed nostrils sometimes accomplishes the object where it had failed by the Politzer method. Sometimes a viscid clot of exudation will stick in the wound in the drum-head and require to be dislodged by the use of a probe, guarded by cotton, or by a pair of bent forceps. You should repeat the leeches daily if the pain does not quickly subside. We have had no experience in such cases in the use of mercurials, and can see no indications for their use. Morphine, with or without bromide of potassium, may be given. Codeine and chloral may also be used to subdue the inflammatory action and relieve pain. The combination of a hypodermic of morphine with a large dose of bromide of potassium internally, seems to meet indications most happily. The best of all laxatives is some form of magnesian salt. The Bitter water given by itself, or with hot water, say a gill or two of the former to a pint of the latter is an admirable laxative. The diet should be nutritive, especially in the earlier part of the day, the occurrence of pain toward night making it inexpedient to fill the stomach at that time.

There is a marked tendency in these acute inflammations of the middle ear for the inflammatory action to become quite brisk in the lining of the external auditory just outside of the drum-head, and especially in the posterior wall of the canal. This seems to be explained by the proximity of the inflammatory focus in the drum cavity, and the contiguity to the back wall of the canal, of the mastoid cells. Acute inflammation of the drum cavity without inflammation, at least of the lining of one or more of the mastoid cells, is of rare occurrence. Of course, there can be no disputing the presence of mastoid cell inflammation when such gross features of it are present as tenderness of, or swelling over, the mastoid process. I do not refer to such cases now, but to those in which inflammatory symptoms occur which, at first, are referable to the drum cavity proper, but soon include that extension of the middle ear in the mastoid cells. When, in the course of a common cold, an ear begins to ring a little, and then, towards nightfall, to become deaf, then painful, and before twenty-four hours have elapsed to relieve itself by a more or less copious discharge, to be followed in a few days by healing and restoration of hearing, I do not suppose the mastoid cells are involved, except in the way of a little hyperæmia of their lining. But when the ear-ache is at all prolonged, and marked by soreness, tenderness, or pain spreading out on the side of the head in a fan-like manner, or darting through the corresponding orbit, or up into the vertex, there is marked mastoid cell disease. Such disease usually gets well—the inflammation resolving; but frequently it leaves tracks, over which it may easily come in recurring attacks, until, at last, a more or less grave osteitis is established. Now, we may lessen greatly this danger by prompt treatment of the very earliest stages of the drum cavity inflammation. I was speaking of the extension of the inflammation to the lining of the external auditory canal. The skin and the periosteum of the external auditory canal are closely incorporated, and when inflammation involves them the resultant swelling is very tender. The moment you see this tender swelling in the external auditory canal make a free incision from a point close to the drum-head outward, as far, if need be, as the ceruminous glands. And if one incision does not divide the inflammatory swelling freely, make one or more parallel with the first. After this, foment by warm douching. This early cutting will give great relief of pain, and perhaps prevent the formation of a carious fistula through the bony wall of the canal into a neighboring mastoid cell. The best instrument with which to make such an incision, is that in fig. 2. The following cases may serve to illustrate what we have just said.

FIG. II.



Case I.—D., æt. 48 ; while using a nasal douche, on the 13th of the month, washed a large dead fly out of his nares. Did not fee any water go up to either ear. Pain, however, started in his left ear soon after. On the 16th I first saw the case. H. D. R., normal ; L. E., 2″ ; tuning-fork heard best in L. E. ; pharynx red and shiny ; left external auditory canal somewhat reddened, and membrana tympani rather red ; Eustachian tube open. Diagnosis :—Inflammation of the middle ear and mastoid cells. Ordered leeches below tragus, warm douche, and paregoric.

17th. Pain relieved, objective symptoms the same.

18th. Pain returned in the night ; repeated leeches and gave hypodermic of morphine, previously incising the membrana tympani below the malleus. A free flow of sero-sanguinolent fluid followed the incision, and was greatly increased by the use of Politzer's inflator. Continued warm douche in external auditory canal.

21st. Politzer used twice a day ; warm douche, three or four times a day ; codeia at night. Leeches repeated on the 20th. On that day the sero-sanguinolent fluid changed to pus, and being more viscid became frequently dammed up in the drum cavity, and was made to flow by douching and politzerizing.

22d. Had a good day yesterday and a bad night. Has had from the first day or so, pain and soreness in head. Took codeia freely, but was kept awake last night by pain in depth of the ear and through corresponding temple ; could not lie on left side of head ; perforation is open ; pus comes freely on Valsalvian inflation, or use of Politzer. Repeated leeching.

29th. No discharge since yesterday; little or no pain; pain returned this morning and has been annoying all day; pain seems more external. Some inflammation of external auditory canal, with swelling, especially of the posterior wall, near drum-head. Ordered leeches again and anodynes.

30th. Was leeched at 9 15 P.M., and again at 11 15 P.M.; took three large doses of paregoric, but awake frequently during the night with pain. Periostitis of back wall of canal worse; made three parallel incisions through periosteum to the bone, one above, one behind, and the other below. Made also another paracentesis of drum-head and evacuated much sero-sanguinolent fluid, aiding the flow with the Politzer, and the Valsalvian method; gave 60 grs. of bromide of potassium in one dose, as there was much pain through the head.

31st. Slept very well through the night.

1st. "Feels more comfortable than at any time since the attack, especially behind the ear." No swelling of mastoid at any time.

2d. The perforation of membrana tympani healed during the night.

3d. Calibre of canal nearly normal; no pain or tenderness over ear or mastoid; no otorrhœa; no trace of wounds of membrana tympani.

6th. Ear perfectly comfortable. H. D. 16".

Went on to full recovery.

Case II.—S., æt. 18. Has had deafness in left ear with offensive otorrhœa for ten years. Complains now of dizziness with diffused pain in left side of head and a tenderness which makes lying on that side disagreeable. H. D. L. E., nails at 3". Tuning-fork, when on forehead, heard best in left ear. Has chronic inflammation of the fauces. External auditory canal filled with offensive pus, on removing which the membrana tympani is found to be red, swollen, and pulsating. There is a small perforation in upper part of membrane, anterior to the malleus. No perforation whistle either from Valsalvian inflation, or use of Politzer's inflator. External auditory canal diminished in calibre throughout inner half and tender to touch.

Diagnosis : Otitis media sup. with mastoid and meningeal complication.

Ord. leech and warm water injection. Treatment interrupted by a malarial attack and a trip abroad.

Sept. 27th. Again examined the ear. No change. Otorrhœa offensive and continuous ; lining of external auditory canal swollen and tender. It being evident that the passage for the pus was indirect, a free incision was now made in lower part of membrana tympani, and by Valsalvian inflation viscid pus was slowly forced out through the opening. This gave a decided sense of relief to the ear.

Dec. 4th. Again saw the case. Still has more or less pain in head ; membrana tympani still reddened and thickened, no trace of the incision ; repeated the latter.

Dec. 9th. The last incision gave comfort in the ear so long as it remained opened ; is now closed ; yesterday had severe headache. To-day made free crucial incision in lower part of membrana tympani but could not get a perforation whistle. Middle ear and Eustachian tube seem to be obstructed by a thickened mucous membrane and viscid pus. Ord. internally potass. iodide ʒij., potass. brom. ʒiij., aq. ℥iv. *M.* Dose, a drachm three times a day. Ear syringed frequently with warm solution of carbonate of soda and a leech from time to time applied inside of tragus.

Dec. 11th. The water when thrown into the ear by syringe now is felt in the throat.

Jan. 4th, 1875. Cannot get any perforation whistle, repeated the paracentesis.

Jan. 5th. H. D. has now increased from ability to hear the click of the finger nails at three inches to watch at one inch and a half and pain in ear and head has subsided.

Jan. 25th. No pain in head since last date. Perforation now open, air whistles through on use of Politzer, but not when Valsalvian inflation is used. Argent. nitrat. gr. x. to aq. distil. ℥i., dropped into external auditory canal and forced through middle ear and Eustachian tube by introducing the nozzle of the Politzer's inflator into the external meatus. Still under treatment.

Case III.—J. C., æt. 37. Took a severe cold in head six weeks ago. Three weeks ago began to have some deafness in both ears, followed by a watery discharge from the right ear and severe pain. The latter did not occur until after the slight discharge alluded to, but immediately became very severe, and after lasting three days was accompanied by a thick, purulent otorrhœa. At the time of the occurrence of the ear symptoms was using a solution of salt and water in a nasal douche and observing every precaution, under medical advice, to avoid strangling and invasion of the middle ears. Had used the nasal douche for many years on the occurrence of nasal catarrh and does not remember to have forced water through Eustachian tubes at any time. H. D. R. E., laid, L. 4″, M. T. R. E. red and somewhat swollen ; deep pain, very severe at night. Did a free paracentesis and ord. leeching. Two days after, ear still painful, swelling of lining of the external auditory canal increased ; otorrhœa continues. Made a free incision through the swelling in the external auditory canal near the membrana tympani, and at the same time repeated the paracentesis.

These were followed by comfortable sleep and an increase of H. D. from watch laid to two inches. The deafness now slowly but steadily lessened in both ears, pain and otorrhœa and all signs of inflammation disappeared.

Case IV.—Mrs. J. N. C. Has had catarrh and been in the habit of using the nasal douche for more than two years. Had never had ear trouble until three or four weeks ago, when her throat became sore and then deafness in right ear with ear-ache followed. The ear-ache has existed "off and on" for the past three or four weeks, and comfort has been promised on the occurrence of "a discharge." Last night the pain attacked the right mastoid region. Left ear normal. The right external auditory canal is uniformly diminished in calibre by a diffused and tender swelling. The right membrana tympani is red and swollen ; H. D., contact ; Eustachian tube is open on the use of Politzer's inflator. The mastoid region is somewhat tender on percussion and shows slight redness and swelling.

Made a free incision of the membrana tympani below and behind the malleus and evacuated a considerable quantity of bloody serum.

On the following day it was noted that the H. D. had increased to two inches, and that there had been no pain during the night. The tenderness in mastoid was also less. This case got well, under observation, in two or three weeks more ; the inflammation showed a tendency twice to recur, but it was easily subdued by two free leechings below the tragus. My purpose is not to give all the details of the progress of this case, but to show the marked effect produced by the free paracentesis. In this case there had been for several weeks an inflammation of the drum cavity, involving the mastoid cells. During most of this period it is fair to presume that the products of the inflammation being serous and not viscid had, in a measure, found their way down the Eustachian tube, to the nares. Slowly, however, the disease was involving more and more of the organ, and would soon have exhibited a profound mastoid cell inflammation, with brain complication. The turning-point in the case from which the cure began was the free paracentesis. Under similar circumstances, I would now not only tap the drumhead, but incise freely the inflamed lining of the external auditory canal.

CASE V.—Feb. 10. G. P., æt. 23. Three days ago began to grow deaf in left ear in church. Tried to relieve the deafness by blowing his nose. Could not. Instilled oil of tar, and almost immediately had pain in the ear. This pain has continued ever since, and yesterday began to outcrop behind the ear, in the mastoid region. Has had a very little bloody discharge from the ear. Assigned cause, cold. H. D. R. E., 2′ 6″, L. 1½″.

Right ear normal in appearance. Left auditory canal shows considerable dried crusts adhering to its walls. Lining grows inflamed and swollen in the vicinity of membrana tympani.

Membrana tympani rather red and swollen, especially behind the malleus. Corresponding mastoid region tender on percussion, but not swollen. Eustachian tube seems to be open on use of Politzer. No perforation whistle. Diagnosis, otitis media acuta. Incised the membrana tympani immediately, and carried a free incision along the posterior wall of the canal. Ordered also a leech below the tragus, and warm water douche.

Next morning, reported "that he had slept all night, and experienced a wonderful difference." No pain, no tenderness over mastoid. No pain on shaking or moving head as before. Has had a bloody discharge from ear all through the night. The swelling in the region of the membrana tympani much less. The Politzer forces out with a perforation whistle a free flow of mucilaginous, viscid, straw-colored fluid.

Feb. 12.—Since yesterday, has had a constant flow of light straw-colored fluid through opening in membrana tympani.

Feb. 13.—Discharge has stopped, followed by some pain. H. D. 2″. No perforation whistle. Examination shows that wound has closed. Again incised the membrana tympani, and ordered a leech below tragus.

Feb. 14.—No discharge or pain since yesterday. Membrana tympani healed. H. D. 2″. Again perforated membrana tympani, and evacuated mucoid fluid, and got a perforation whistle. The case went on favorably until the 18th, four days, when the external auditory canal began to be invaded by a very painful swelling, of a diffused, furuncular character. Ordered quinine and cod-liver oil, and leeches.

Feb. 22.—Incised a small abscess in the floor of the canal.

March 8.—H. D. 2′ 6″. No evidence of disease except slight redness of the membrana tympani, and adjacent lining of the canal. After use of Politzer, H. D. increased to 3′ 6″. To continue cod-liver oil.

Under similar circumstances, I would now, in addition to the remedies employed on the 18th Feb., in the above case, make a free incision through the inflammatory swelling of the canal.

We cannot be too prompt in evacuating the middle ear in cases of inflammation of that part. By delay we can gain nothing, as the paracentesis never does any harm. And the beneficial effect of an early incision, made through an inflammatory swelling in the external auditory canal, often extends to the more remote seat or focus of the inflammation in the middle ear. I wish that I could impress upon you the importance of applying these principles in the treatment of the ear complications of measles and scarlet fever. You should invariably examine the ears *daily*, in all cases of measles or scarlet fever, and anticipate, if possible, that ulceration of the membr. tympan. which so frequently occurs in the progress of those maladies. A vast number of cases of obstinate or incurable otorrhœa and chronic middle-ear disease might be prevented if the general practitioner would scrutinize daily the ears in those cases in which ear complications are likely to occur. And of all the remedies employed in such cases paracentesis of the drum-head is the most valuable. No practitioner is really prepared to treat a case of measles or scarlet fever who is not able, at east, to recognize the drum-head when he sees it, and to perform the simple operation of paracentesis. Again I remind you that my intention, in this colloquial lecture, is not to teach experts, but to help general practitioners to apply some of the principles of hygiene and surgery to the very large and common classes of catarrhal and other inflammatory affections of the organ of hearing.

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CAPILLARY BRONCHITIS OF ADULTS; THE FREQUENCY OF ITS COMPLICATION WITH CHANGES IN THE AIR-VESICLES, OR PARENCHYMA OF THE LUNGS, AND ITS RELATIONS TO CATARRHAL PNEUMONIA.

BY

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I INTEND only to speak of the disease as it appears in adults, as, in young children and aged persons. it presents certain peculiarities which require separate mention.

The object in bringing this subject before you is to show that the descriptions of capillary bronchitis ordinarily given are of such a character as to create much confusion, and throw doubt over a really correct diagnosis. I shall endeavor to prove that the lines between this disease and catarrhal pneumonia are too sharply drawn ; that its complications are often so entirely unnoticed, or so obscurely hinted at, as to make it impossible for those who have not had special opportunities, and particularly those who are just beginning their studies, to recognize the affection. Wishing to call your attention to certain points in connection with diagnosis alone, I shall not allude to the treatment which you have had an opportunity to observe.

During the past winter the unusual inclemency of the weather brought to the hospital quite a number of cases which furnish material to illustrate many important points in connection with capillary bronchitis, which is generally regarded as a rare disease, and which certainly is so, if we are to be guided by the descriptions found in the usual text-books. You have seen the cases in the order in which they have presented themselves, and it will now be profitable to review them as a group, so arranged as to bring out their striking characteristics still more strongly.

I shall first bring up such as resemble most closely those of pure capillary bronchitis, and show that even these give evidence of something more than a catarrh of the smaller air-passages, and that the complications are owing to an affection of the air-vesicles or pulmonary tissue; and, finally, such as might be classed under the head of catarrhal pneumonia, in which the deeper seated changes are as prominent, or more so, than those of the air-passages.

In weighing the cases to be brought before you, endeavor to free your minds, as far as possible, from all preconceived ideas connected with the ordinary nomenclature of special diseases of the lungs. Take the facts as they present themselves, interpret them in accordance with your general knowledge, and, above all things, remember that I intend in no way to allude to tubercle.

We will talk about things, about processes, and, after we have arrived at our conclusions, we will show how the terms in general use will apply to these processes.

CASE I.—An Italian, æt. 22, entered the Massachusetts General Hospital, on December 13th, 1874. He could not speak English, and, as there was no interpreter, we were thrown upon our own resources for the elucidation of the case.

The expression of the countenance was good, the color of the face natural. The cough was frequent and accompanied by abundant muco-purulent expectoration. The respiration was 28; the pulse 120; the temperature 103.6°.

The quick pulse and the high temperature indicated a febrile disease. The absence of emaciation, or of other signs of interference with the nutrition, led us to suspect that this disease was of an acute rather than a chronic character. The accelerated respiration pointed to the chest as the seat of the disease. There was no indication of pain which called attention to any particular part of the chest. There was no attempt to suppress the severe cough, as is usually the case when such pain is present as we meet with in inflammatory affections of the pleura. We should, therefore, rather suspect the air-passages. This suspicion is made still stronger by the character of the expectoration, which consisted of somewhat purulent mucus, so viscid that it could be spread out in water as a continuous mass or layer. But the urgent dyspnœa and the marked constitutional signs made it probable that the larger air-passages were not alone affected. Such urgent symptoms might be caused either by an affection of the heart, of the smaller air-passages, or even the air-vesicles, or by some complication which the symptoms did not indicate. The quick pulse and high temperature pointed to something more than simple asthma.

It was, therefore, necessary to resort to an examination of the chest, before giving a positive opinion. The resonance was normal in all parts, except in the lower sub-axillary region on the left side, where the pitch was somewhat higher. This higher pitch was owing either to some change in the parietes, to the interposition of some fluid or solid material between the parietes and the lung, or to a change in the lung itself. Nothing was noticed in the parietes, and any change which would cause such a difference on percussion is rare, unless connected with some former attack of pleurisy of which we have no knowledge. Taking into consideration the situation and indefinite outline of the dullness, we should rather suspect that the lung was the seat of the difficulty. But we could not determine this point without resorting to auscultation. This showed, with normal heart-sounds, the presence of subcrepitant râles in all parts of the chest, with perhaps a few coarse moist râles. The moist character of the râles showed that air was mingling with fluids in cavities or canals, of which the size varied with the size of the râles, the few coarse ones indicating larger spaces than the numerous fine ones. Now the latter were not such as are heard in cavities formed by the destruction of the pulmonary tissue. They were not only finer, but their uniformity and universality were incompatible with the supposition that they occupied the seat of destroyed tissue. They could only have their seat in the uniform natural channels ; they were, in a word, precisely what daily experience leads us to associate with the smaller air-passages. The statement that the fine moist râles are formed in the smaller air-passages is in accordance with the generally accepted belief, which, also, sanctions the locating of fine dry râles in the air-vesicles. Though not able to prove to you that the line between the air-passages and air-vesicles can be so absolutely drawn by physical signs, though recognizing the difficulty of distinguishing absolutely, in all cases, between the fine moist and dry râles, there is such an approximative truth in the statement as to warrant us in accepting it as a basis for diagnosis without repeating the above qualification in the cases which follow.

Our next inquiry is in regard to the nature of this more or less fluid material, and its source. It might be blood which had escaped from the bloodvessels at the points where the râles were heard, or it might have been drawn into the bronchi from a single remote source. It might have been serum, such as is found in œdema, or a product of the mucous membrane itself, consisting of mucus, or muco-purulent matter. Now, we could not conclude that this fluid was blood, for the râles were too general, and there were no other indications of hemorrhage. The absence of œdema elsewhere, the evidence we had already obtained of acute febrile disease, with such marked thoracic symptoms, made it probable that disease of the bronchi themselves had been followed by an increased formation of liquid, of the character of which the expectoration gives us very clear evidence. But, as there are exceptional cases, in which œdema of the lungs may be the first indication of disease of the kidneys, the urine should have been examined before excluding this. Still, without such an examination we could conclude, with but little risk of error, that the smaller air-passages were the seat of the disease, and that this disease was of an inflammatory character.

But we have not explained the higher pitch, shown by percussion of the left lower sub-axillary region. Though some old affection of the pleura might give rise to this difference, we had no knowledge of such, and, having already ascertained that the bronchi were the seat of disease, it was reasonable to infer that the pulmonary tissue itself might have undergone some change which would explain the morbid sign, though not sufficient to give us farther evidence. We know that, when the smaller air-passages are inflamed, the pulmonary tissue is liable to be involved more or less. Portions, often small, but sometimes quite large in the aggregate, may be deprived of air, other portions may become œdematous, and others may be solidified by the accumulation of mucus, serum, and cells in the air-vesicles. We have reason to fear that all of these conditions may have been present to such an extent as to give rise to a variation of pitch, though not sufficient to solidify a large and continuous portion of lung. Consequently, we had no bronchial respiration and no modification of the voice.

PROGNOSIS.—Though, without the history of the case previous to the time when the patient entered the hospital, we were obliged to form as complete an opinion as possible about the result. Bearing in mind the character of the disease and its gravity, we could but express our fears of a fatal termination, but, as there were no special signs to indicate this, and as recovery is not uncommon, we did not give up hope.

For some days, the cough and expectoration continued very abundant, but, on Dec. 22d (nine days after entrance) both were reported as less. At the same time, the subcrepitant râles, though still heard at the lower part of both sides of the chest, were much less evident. The pulse gradually fell, and, on the 21st, was reported as 84. The temperature, two days before, had fallen to the normal point, and, on the 24th, the patient felt so much better he left his bed, dressed himself, walked away from the hospital, and resumed his occupation of fruit selling. He gained in flesh and his cough disappeared during the following two months, at the end of which time he disappeared.

At the time he left the hospital the principal feature which excited doubt, was the dullness in the lower part of the left side of the chest, of the significance of which future cases may furnish proof.

Still, the most marked features of the case were the dyspnœa and the fine moist râles, indicating an affection of the smaller bronchi, and, inasmuch as the general symptoms became less marked as these physical signs diminished, we are justified in connecting the two.

Case II.—On Jan. 27, 1875, an Irish laborer, 30 years old, entered the Massachusetts General Hospital. He reported that he had lost one brother by consumption, but had himself never been ill before. Two weeks previous, without known cause, he began to cough and expectorate considerable yellow matter, but there was no pain in any part of the chest. About a week afterwards dyspnœa was first noticed, and soon became very marked on slight exertion. At the same time his voice become husky. These symptoms persisted, and, when he was first seen, were more marked than ever before. He reported that he had had no chill and only occasional feverish feelings. The appetite had been very bad, and the tongue was covered with a thick whitish coat. The bowels were regular. P. 112. Temperature 102.8°.

The symptoms had been so severe as to confine the patient to the bed the greater part of the time since the beginning of the attack. The sleep had been disturbed by dyspnœa and cough.

The attack was evidently acute. The high temperature and quick pulse indicated a febrile disease. The dyspnœa was very marked, the muscles of the neck were contracting strongly, and the cyanotic hue of the face showed how much the aeration of the blood was interfered with.

These signs, with the cough and expectoration, again called our attention to the chest. The previous good health of the patient made it very improbable that any chronic disease of the heart would be found, and the symptoms certainly did not point directly to this organ as the seat of acute disease, though such was possible.

The absence of any pain, the severe cough and the abundant muco-purulent expectoration, led us to suspect some disease of the air-passages, while the great dyspnœa indicated that the smaller air-passages were affected; but, without the physical signs, we could only conjecture.

Those of the heart were normal. There was well-marked though ill-defined dullness or a higher pitch in quite an extensive portion of the middle region of the posterior part of the right side of the chest. For reasons given in the previous case, we connected this dullness with some change in the density of the lung itself, but the absence of pain and the character of the expectoration did not indicate an ordinary attack of croupous pneumonia, but this could not be absolutely excluded without auscultation. The latter showed subcrepitant râles with some of a sonorous and sibilant character over the greater part of the chest, increasing with the disease, from the apex.

Though there was nothing in the history of the case which suggested disease of the kidneys, as a cause of œdema which might give rise to subcrepitant râles, it was thought best to examine the urine and be sure that there was no complication of the kind. The excretion was found normal. This source of error being eliminated, we may say that the physical signs confirmed our suspicions that the smaller air-passages were the seat of the trouble. In addition to this, for the reasons given in the preceding case, we concluded that there was other disease in the right lung, which involved the air-vesicles, or the parenchyma.

For a few days there was no material change in the symptoms, but by Feb. 3d slight signs of amendment were noticed. By the 8th the patient reported himself "first rate," and on the 9th he was up and dressed, though the cyanotic hue of the face was still noticeable and there was evidently much difficulty in breathing. Still, the improvement in his general appearance and strength was constant. The cough, however, continued very troublesome, and the expectoration abundant, though by Feb. 12th it was reported as less so. The appetite also improved. The pulse diminished in frequency. Being 108 at the beginning, it fell, after some fluctuation, to 72 on Feb. 10th, though it rose slightly afterwards.

The temperature also fell, so that by Feb. 7th it was 99° in the evening, and it rose but a few tenths of a degree above this at any time afterwards. The voice continued hoarse, and an attack of tonsillitis, on Feb. 20th, caused much constitutional disturbance, which lasted four or five days.

The improvement in the physical signs kept pace with that of the general symptoms. On Feb. 7th, eleven days after entrance, there was noticed a decided diminution, almost absence of sub-crepitant râles in the upper part of the chest, but the dullness still persisted in the right side. On the 18th the râles were less moist than before, and the dullness had diminished very much. On the 24th dullness was still noticed and a few sonorous, sibilant, and sub-crepitant râles were heard in various parts of the chest, but particularly in the right side. These râles suggested the idea of air forced through narrow passages, the inner surfaces of which were covered with a viscid material. They were most marked in expiration, of which they occupied the whole period. There was also a slight-friction sound in the lower part of the right side of the chest posteriorly.

This case resembles the first very closely, but the dullness was much more marked, and we could speak much more strongly of the condensation of that portion of the lung. The danger from this source was much greater than in the previous case, as the products of inflammation within the air-vesicles were liable to undergo caseous degeneration, and the pulmonary tissue itself might be destroyed. Still the diminution of dullness was favorable, as was also the general improvement, which kept pace with the diminution of the physical signs. While in this condition, in the latter part of February, the patient left the hospital, but was seen by Mr. Whitney on April 15th, from whom the following additional facts were obtained: For a few days after leaving the hospital the man attempted to work, but was obliged to desist on account of dyspnœa and weakness. On April 1st, he again resumed work and has been able to continue, though he still complains of dyspnœa on exertion, and of paroxysms of cough, without expectoration, three or four times during the day. The cough is so severe as to cause him to vomit at times. The appetite is still poor, but he thinks he has gained flesh. He sweats on slight exertion, but not, as formerly, while quiet. He had not noticed any lividity of the face within a short time. The chest was everywhere resonant and the respiration vesicular without râles.

Case III.—A Norwegian laborer, æt. 34, entered the Massachusetts General Hospital December 24th, 1874. His family history was good, and he had never been ill before. Four weeks previous he was exposed to wet and cold. On the following day he was obliged to give up work, as he felt chilly and began to cough. After remaining idle for two days he returned to his work, but was not able to do as much as before. Not long after, dyspnœa came on, and increased so that he took to his bed twelve days before entrance. Dyspnœa and cough had been the prominent symptoms, and both were so much aggravated by lying down as to make the sitting posture preferable. The cough was so severe as to excite vomiting at times, and was accompanied by abundant muco-purulent expectoration. He had been more or less feverish, and, a few days after entrance, the temperature rose to 101° in the evening. The pulse was upwards of 100. The appetite had been poor, the bowels constipated. The urine was high-colored, but not remarkable in other respects.

The history was that of an acute febrile attack, and the symptoms pointed to the chest as the seat of the trouble. The face wore an anxious look, was slightly livid, and bathed in perspiration.

The consideration of the rational signs and the character of the expectoration indicated, as in the preceding cases, that the smaller air-passages were the parts principally affected. But the testimony of the physical signs was still stronger.

There was little movement of the chest, while that of the abdomen was exaggerated, and the scaleni muscles were acting powerfully. Percussion showed, perhaps, a little dullness towards the base of the left lung behind and on the side, but the dullness was quite well marked on the right side, in front, from the third rib downwards. As in the preceding cases we considered this dullness as indicative of some new-formation in the air-vesicles or pulmonary parenchyma, but auscultation was necessary to determine the matter. Sibilant and sonorous râles were heard in all parts of the chest, both in inspiration and expiration. In addition to these, there were in the lower part of the left lung others more moist in character, like subcrepitant râles. Still they were mainly sibilant or sonorous, and differed in this respect from those in the other cases. They were such as are supposed to be caused either by the passage of air through tubes with swollen lining membranes, or containing thick mucus. The sound in the lower part of the left side also resembled that caused by the rubbing of inflamed surfaces against each other. We were sure in this case of the presence of a large amount of muco-purulent liquid, as it was expectorated ; but for some reason, although the bronchial secretion resembled that of the other cases, the râles were mostly dry. Though it was impossible to explain in words their exact character, they gave the impression that many of them had their seat in the smaller air-passages, where we must certainly locate the moist ones; and if we take into consideration the marked dyspnœa, which is not explained by the amount of the deeper-seated disease indicated by the limited dullness, we must suppose that the smaller air passages are very extensively affected.

But how shall we explain the marked dullness or flatness over the anterior part of the right lower, and the antero-lateral portions of the left lower lobes? We had no physical signs here which were not found elsewhere, except the suspicious friction-sound on the left side; there was no bronchial respiration no increased resonance of voice, no sign which we expect to meet with in cases where there is consolidation of the lung. Still, as we had dullness on percussion, we may perhaps explain it, as in the preceding cases, by appealing to the well-known fact that when the smaller air-passages are the seat of inflammation the air-vesicles are liable to become affected in such a way that the density of the tissue is increased. But the parts thus changed may be so separated from the larger bronchi by aerated tissue, as to prevent any conduction of sound from them. We therefore concluded, that the rational and physical signs showed inflammation of the smaller air-passages and condensation of certain portions of the pulmonary tissue.

The prognosis was unfavorable. The character and amount of the disease were such as to make it very improbable that the patient in his debilitated condition could offer a long resistance.

The fears of a fatal termination were soon verified. The cough continued very troublesome; the expectoration became more and more purulent; the dyspnœa, with some short intervals of relief, obliged the patient to remain in the sitting posture most of the time; the perspiration was constant and profuse. The appetite was poor, and the tongue was covered with a thick brownish-white coat. The pulse continued high, and the day before his death rose to 120; the temperature continued at about 100°. The urine was high-colored and had a specific gravity of about 1.025, but was in other respects normal.

The physical signs did not change materially, though subcrepitant râles were at times noticed in the left side. A few days before death the feet swelled, but this swelling never became very marked. He gradually grew weaker, and died on Jan. 13th, seven weeks after the beginning of the attack.

The autopsy was made twenty-one hours after death. Dr. R. H. Fitz furnished the following report of the appearances: The pleural surface over the lower part of the lungs was covered with a dense yellowish-white false membrane.

The anterior and lower third of the left upper lobe, as well as the anterior half of the right lung, were dense, distended, and non-crepitant. These portions were more especially covered with the false membrane previously referred to.

On section the upper parts of the lungs were crepitant, reddened, not particularly œdematous. The lower parts in general were pale gray and translucent, the lobules being very distinctly marked. The bronchi of these portions contained yellow opaque fluid, of the consistency of cream, which was readily squeezed from the smaller bronchi. The upper border of this infiltrated portion of lung presented small patches about the size of beans, dotted with yellow, containing in their centres bronchi with more or less purulent secretion, in relation to which the infiltration of the surrounding tissue was very direct. The portions between these broncho-pneumonic nodules and the denser infiltration of the lung tissue. were of a reddish color, moderately dense, and somewhat œdematous. Further examination of the gray infiltration at the lower part of the lung showed, very generally, exceedingly minute yellow points, quite opaque, apparently owing to fatty degeneration of the alveolar contents, and large yellow opaque spots and lines suggesting bronchial canals. The larger bronchi contained an abundant purulent secretion, and the mucous membrane was reddened. The bronchial glands were considerably enlarged, gray, and soft.

The aorta was much diseased, but as the changes were chronic and had no bearing upon the points we are endeavoring to make clear, they need no further notice.

Dr. Fitz also made the following report of the microscopic appearances : "The examination of sections from the hardened lung showed the existence of recent and chronic changes. In certain parts the alveoli contained masses of coagulated fibrin and cells, such as are constant in croupous pneumonia. This condition was a comparatively limited one ; more common was the presence of large corpuscles often containing pigment, others of an epithelial character with rarer red blood-corpuscles and the small round indifferent cells. Such accumulations were generally imbedded in a finely granular material (coagulated albumen). Portions of the lungs were found to be densely filled with small round cells to such a degree that the alveolar walls were often made out with difficulty. In other places, and this condition was very extended, the alveolar walls were thickened and their cavities apparently filled with a connective tissue of a reticulated character, in which capillary bloodvessels were readily observed. In many places the interstitial tissue of the lungs was infiltrated with small round cells, and rarely the larger bloodvessels were found to contain thrombi composed mainly of white corpuscles, though not entirely so.

"Though the gross appearances indicated the existence of granular corpuscles in abundance, the sections having been treated with alcohol, the characteristic appearances of such no longer existed.

"To summarize, the lung may be regarded as presenting alterations due to croupous pneumonia, catarrhal pneumonia, desquamative pneumonia, and interstitial pneumonia, the latter more especially of a chronic character."

This case is particularly interesting, as the evidences of consolidation of the lung were quite marked during life, and as the autopsy furnished us an opportunity to know upon what pathological changes this consolidation depended. The condition of the external surface of the lung showed that some of the sounds which it was supposed might be caused by the movement of opposed inflamed surfaces could certainly be explained in that way. The pus, forced from the minute air-passages, showed that they were also the seat of inflammation. We have, finally, the most unmistakable evidence that the dullness of certain portions was connected with the appearance of morbid products in the air-vesicles themselves.

The microscopic character of these changes is of the highest importance, in connection with the present inquiry. The result of this independent examination, made by a competent observer, gives us the final proof of that combination of processes which it is our object to make clear, and which will become still more apparent after the examination of the cases which follow.

The latter differ materially from those already reported, in the extent of the bronchial inflammation.

Case IV.—On Nov. 18, 1874, I visited a young man 16 years old, who, on the 14th, after active exercise, by which he was much heated, rode in an open wagon nine miles and was much chilled. The next night he had a chill, followed by fever. There was but little cough till the night of the 17th, when it kept him awake.

When the patient was first seen he was up and dressed. He had even attempted to go out, but was so faint he was obliged to remain at home. He complained principally of dizziness. The pulse was 96, the temperature 102°. There was no appetite.

The constitutional symptoms indicated an acute disease, which the cough led us to locate in the chest. The general symptoms were less marked than in the ordinary inflammation of the pleuræ or lungs, and both the pain and expectoration, which generally accompany such diseases, were wanting. A severe bronchitis would have explained everything, but it was impossible to be sure that a limited portion of the lung had not been attacked by more serious disease, as such will sometimes cause much constitutional disturbance.

On examination of the chest nothing was noticed except a transient doubtful subcrepitant râle, at about the second or third rib on the right side in front. There was no change of resonance.

This sign, slight as it was, if it indicated anything, indicated the presence of a fluid material in the smaller bronchi of that limited region. The question at once arises whether the amount of disease announced by these very limited and doubtful sounds was sufficient to explain the marked constitutional symptoms. This may seem improbable, but experience teaches us that a very limited amount of disease in certain parts of the body may produce much greater effects than would be anticipated. A sore throat, apparently slight, may cause more constitutional disturbance than we notice in our patient. Still, we must recognize the possibility that a deep-seated disease might be covered up by healthy lung, so that no change in resonance would be detected, and the auscultatory signs might be very obscure. The presence of serious disease was feared, and the necessary precautions were taken. The patient was confined to the house, and strict directions were given to guard him against changes of temperature or other bad influences.

The dizziness, which was the first prominent symptom complained of, diminished gradually, and, by Dec. 2d, was noticed only on running. The pulse fell on the 19th to the normal standard, and on the 22d was as low as 56, and only once afterwards became unnaturally quick, perhaps from accidental causes. The temperature also fell in a week to the normal point, and scarcely varied from this afterwards. The appetite improved, and on Dec. 8th was as good as ever. There was no pain at any time, except some at the base of the chest, caused by cough. The latter, which was at first urgent, gradually diminished until it disappeared with the other symptoms.

For several days after the first examination, only a few doubtful moist râles were heard in the right side of the chest, near the second rib, and, perhaps, in other parts of that side of the chest, but no dullness on percussion was ever noticed. On the 28th, however, well-marked subcrepitant râles were heard over the right upper lobe, between the clavicle and the third or fourth rib. These gradually diminished, and, on Dec. 6th, but a trace of them was heard at the end of expiration, and, on the 8th, the respiration was perfectly normal, and the patient considered himself well.

My object in presenting this case has been to show you that the same kind of râles which we have previously heard over the whole chest may be confined to a very small portion of it, may be the only positive sign, and yet may be accompanied by marked constitutional symptoms. In whatever way we explain their absence at first, or their subsequent appearance, they were of precisely the same character as those which were more widely disseminated; the difference was only in extent. In all the cases they indicated the same lesion, viz.: an inflammation of the smaller air-passages.

But let us suppose that the circumstances of the patient had been such that further exposure was inevitable; that the gravity of the disease, as shown by its seat and character, had not been foreseen, or that there had been some constitutional predisposition to pulmonary disease, the result might have been very different, as the following case will show:

Case V.—A stone-cutter, æt. 21, entered the Massachusetts General Hospital, on October 9th, 1873. He reported that his parents were healthy, and that he had always enjoyed good health until one month before entrance, when, without known exposure, a cough came on and had persisted, accompanied by scanty expectoration, but no hæmoptysis. There had been considerable dyspnœa on exertion, and he had had night-sweats with loss of weight and strength. Still, he continued to work until nine days before entrance, when he took to his bed and remained there; at the same time, he began to have pain in the left side of the chest on taking a full breath or lying on the right side. The appetite had been pretty good; the bowels had been so much constipated as to require the use of laxatives. Nothing abnormal had been noticed in the character of the urine, or in its passage. Pulse 98; temperature 103°.

The general symptoms indicated a subacute febrile attack, and pointed to the chest as the seat of the disease. The series of symptoms was much more complex, and differed materially from those of the preceding cases. The duration of the disease, its slowly progressive character, the absence of excessive dyspnœa and pain, led us to infer that the result of a complete examination would differ from that arrived at before. It was not necessary to dwell upon pleurodynia or other painful affections of the walls; we had certainly something deeper. The pain suggested some inflammatory affection of the pleura, but the cough and expectoration indicated some disease which involved the air-passages, and probably the pulmonary tissue also. The supposition that the mucous membrane of the bronchi was alone inflamed would not explain the decided constitutional symptoms, unless the smaller air-passages were involved, and the pain implied something more.

An ordinary inflammation of the lung is ushered in by more marked symptoms, and, unless the disease is very limited, prevents the patient from keeping about. On the contrary an inflammatory affection in which the smaller air-passages and vesicles are involved to a limited extent, or progressively, may begin insidiously, may not be sufficiently severe to prevent work or seriously interfere with it, and may at any time, by its increase, give rise to such symptoms as were noticed in this case. We have, therefore, reason for believing that both the bronchi and pulmonary tissue of some part of the left lung are involved.

This view, though very probable, it was necessary to confirm by an examination of the chest. There was found dullness, beginning at the fourth rib outside of the cardiac region, and extending under the axilla to the spine of the scapula, and thence nearly to the base. This dullness could be attributed either to some solid or fluid interposed between the parietes and the lung, or to some change in the lung itself.

The form and position of the dullness were not such as are generally seen when there is fluid in the pleural cavity, the dullness in that case generally beginning below and extending upwards, though adhesions may confine the fluid to any portion of the cavity. A large tumor might have caused flatness in the region described, but such a growth is very rare, and a supposition that it might be present was not sustained by the rational signs. The form of the dullness, with the expectoration and general symptoms, indicated rather an affection of the lung.

On auscultation, bronchial respiration and bronchophony were heard in front and on the side, with crepitant and subcrepitant râles; the latter were also heard in the back. The bronchial respiration showed the presence of some medium with a greater conducting power than the healthy lung, between the ear and the original seat of the sound. Such might have been an effusion, which is, also, often announced by an increased resonance of voice, but the râles made it evident that the lung itself was beneath the ear. After considering the various causes of these râles we came to the same conclusion as before, that they were owing to a morbid formation of fluid materials in the smaller air-passages. Knowing how frequently the air-vesicles are affected, when the smaller bronchi are the seat of disease, we were prepared to accept the testimony of the bronchial respiration and bronchophony as indicative of consolidation. The fine crepitus left no room for doubt. It is the sound which is especially associated with inflammation of the air-vesicles, and, extending as it did beyond the confines of the solidified portion, it announced a progressive inflammatory disease. Then the fears based upon the rational signs were confirmed, and the subsequent course of the case verified the grave prognosis.

On October 22d, twelve days after the above record was made, solidification of the lung had so far increased that the bronchial respiration was heard in the left back near the lower angle of the scapula, and fine moist (subcrepitant) râles were heard over the whole lower part of the back.

While the disease was making progress here, the right lung was also invaded, and, on October 31st, distinct crepitant and subcrepitant râles were detected near the lower angle of the right scapula, where there was also some questionable dullness. We were sure that these signs were new, as pain had led us to examine this portion of the chest several days before they appeared. We have, therefore, watched the development of disease upon the right side, similar to that with which the patient entered the house.

On November 5th there was dullness on percussion over the lower and posterior half of the right side of the chest, with fine crepitant râles outside of the lower angle of the scapula, and bronchial respiration and increased resonance of the voice inside and below the lower angle of the scapula. Three days after, the physical signs became still more marked, bronchial respiration, bronchophony, and crepitant râles being heard below the fifth rib in front.

On December 5th many somewhat coarse mucous râles were heard over the dull region on the left side, while those of a finer character were still heard in the back, with the cough. On January 9th the healthy respiration had diminished, the moist râles had become coarser and more liquid, while the crepitant and subcrepitant râles, which characterized the earlier stages, were heard on coughing.

On February 3d the dullness persisted everywhere with a kind of tympanitic resonance over the lower part of the left side, in front, where there was gurgling with amphoric respiration. There were also coarse liquid râles in other affected portions of both sides.

Through these physical signs I have traced the course of the disease from its beginning until it terminated in the destruction of the pulmonary tissue, for the latter was surely announced by the gurgling and amphoric respiration—both of which showed the presence of cavities.

While these changes were going on in the lungs, the constitutional symptoms were very marked. Though he appeared somewhat better about the middle of October, this change was only temporary. Pain in both sides of the chest was so severe as to require the frequent use of morphine, and the application of a blister. The cough was generally very troublesome, though there were intervals of relief. The expectoration was generally abundant and muco-purulent, with a trace of blood at one time. There was always more or less dyspnœa, but it was never so extreme as in the earlier cases reported. Chills and perspiration were frequently complained of. The pulse varied from 80 to 100 during the first month, and then, during the rapid development of the disease, rose as high as 116–120, but again fell and showed a lower average, until towards the close, when it rose rapidly. The temperature showed peculiar variations, being often reported as normal in the morning, while it rose to 102° or 103° at night. The appetite was very deficient. The emaciation became more and more marked, the debility gradually increased until he became very feeble, and he died on February 8th. The autopsy was made by Dr. R. H. Fitz. "Both lungs were extensively infiltrated with cheesy material, and contained cavities of considerable size at the base as well as the tops. In addition, nodules and granules, gray and opaque, of the size of a pin's head, many of which, particularly the larger, presented the appearance of peribronchitis, while the smaller might have been miliary tubercles. The spleen was large, anæmic, the pulp increased, and the follicles distinct. The kidneys were enlarged, particularly the cortex. The capsule was readily detached. The surfaces were irregularly mottled with peculiar yellow specks, corresponding with the region of the convoluted tubes. The liver was enlarged and contained several minute gray points—apparently miliary tubercles. The stomach and intestines presented a moderate degree of catarrh."

If we contrast these appearances with those described in connection with the third case we see a marked difference in the extent of the disease, and some additional features. The large masses of caseous matter showed a degeneration of the morbid product, the gradual formation of which we watched. The destruction of the pulmonary tissue was such as is frequently met with in cases of the kind. In regard to the doubtful tubercles they may have formed in the last stages of the disease, but were indicated by no signs and have no special interest for us. The other organs presented such secondary changes as are common in inflammatory affections.

These few cases are not brought up as sufficiently numerous to prove anything, but merely as examples of what may be met with in practice, and to show by their variety the points of correspondence in diseases, which have received different names. Though introduced under the head of capillary bronchitis, I have carefully avoided the subsequent use of that term, as I wished to talk about processes rather than names, until we had become convinced of the character of these processes, and of the various conditions under which they might occur. It is now time to recapitulate and see whether we can properly class our cases under the head of capillary bronchitis alone. or whether some other name would be more applicable.

We have in all of them detected the same kind of fine moist râles which are admitted to have their seat in the smaller air-passages. We showed clearly that the moisture to which these râles were due was connected with an inflammation of these air-passages.

Judged from this symptom alone, the first three cases correspond with the description of capillary bronchitis given by all writers. but, in each of them there was dullness on percussion over certain regions, which would place them in the category of exceptional cases. But, even if the difficulty were removed, the discrepancies between the accounts of different writers are such as to still leave us in doubt whether they intended to refer to similar cases.

Let us begin with the description of Valleix,* who quotes largely from Fauvel, a name everywhere associated with capillary bronchitis. He says, "The causes are those of ordinary bronchitis, and the disease begins as a simple bronchitis, but extends to the greater part of the extremities of the bronchi. The gravity is in direct relation to the extent of the inflammation, to which also its peculiarities are also due. Percussion gives negative results, and we often have increased resonance." According to Laennec, towards the approach of death, there may be diminution of resonance near the base, owing to serous or sanguine congestion but Valleix does not describe these conditions in connection with capillary bronchitis, as he attributes the dullness to œdema, or some other mortal disease of which the bronchitis is only a complication. Fauvel distinguishes the disease from lobular pneumonia by the more intense dyspnœa, the great anxiety, the extent of the râles in the chest, and by the symptoms of asphyxia, which are much less marked in lobular pneumonia at the beginning. The disease is always very grave, and almost always fatal.

After death, in addition to the muco-purulent fluid in the bronchi, there are found in the majority of patients purulent granulations disseminated about the lung, particularly at the surface of the inferior lobe.

Subcrepitant râles are spoken of as the principal physical signs. but resonance on percussion is strongly insisted upon, while the dullness on percussion is thrown out. Still, in speaking of the morbid appearances, purulent granulations are described as disseminated about the lung, particularly at the surface of the inferior lobe. Do not the latter show something more than bronchitis?

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* *Guide du médecin praticien.* Paris, 1851.

If we appeal to Niemeyer * our difficulties only increase. In direct contradiction to the previous description, we are told that, though there is dyspnœa it very rarely reaches a point to produce a sense of suffocation. The patient always succeeds in introducing air into the alveoli. Sometimes, however, there are paroxysms of dyspnœa, which show that the vagus is involved, and that there is a contraction of the muscles of the bronchi. Percussion may be as little changed as in catarrh of the larger bronchi. In partial thickening of the mucous membrane, instead of sonorous, we may have sibilant râles, and when the secretion is abundant subcrepitant ones. If we have pain it is an indication that there is a complication; but we may have pain caused by cough at the point of attachment of the muscles of the abdomen to the chest. The disease is rarely accompanied by danger, and generally ends in one or two weeks.

Though the description corresponds to a certain extent with the cases we have seen, and those described by Fauvel and Valleix, nothing is said about the liability to complication with disease of the pulmonary tissue, and the declaration that the disease is rarely accompanied by danger will certainly not apply to what is ordinarily called capillary bronchitis. The only difference between this and ordinary bronchitis seems to be, that, in the former, the smaller air-passages are affected instead of the larger ones, and we are taught to expect that the disease will pass off in the same way.

Dr. Austin Flint † tells us that "the affection has but recently come to be understood. It differs widely from ordinary acute

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* *Lehrbuch der speciellen Pathologie und Therapie.* Berlin, 1866, p. 75.

† Practice of Medicine, p. 207; Phila., 1868.

bronchitis, as regards its clinical history and danger. It is an exceedingly grave affection, proving fatal, especially when it attacks young children, in a large majority of cases. The danger proceeds from the obstruction to the current of air to and from the air-vesicles." "Although the inflammation reaches so near the bronchioles and air cells it does not tend to extend into the latter; in other words, capillary bronchitis does not eventuate in pneumonitis. Cases in which these two affections become combined, if they occur, are rare exceptions to the rule. The resonance on percussion is not diminished in capillary bronchitis, and it may be greater than in health owing to an emphysematous condition of the air-vesicles." "As the affection is bilateral, these moist bronchial râles produced in the smaller tubes, and those of larger size are heard on both sides, and, especially, on the posterior aspect of the chest."

The same author, in his work on "Diseases of the Respiratory Organs,"* states that "undiminished resonance on percussion on the two sides, although negative, is a fundamental point in the diagnosis. The dullness denotes either that the affection is complicated with pneumonitis, or that a certain amount of collapse has taken place."

The great danger is very properly insisted upon, but, if it be true that the disease does not tend to extend to the air-cells, we should be obliged to consider our cases as exceptional. The admission that it may end in pneumonitis is certainly important, but there is reason to think that the danger is much underrated. The restriction of the disease to the air-passages is insisted upon very strongly. Technically, we may assume that anything more than bronchitis is a complication, but, practically, and clinically, we must bear in mind that so sharp a line is not drawn by nature.

Thus far I have only quoted writers who, not only, by their contradictory statements, would leave you in doubt as to what constituted capillary bronchitis, but who would give you good reason for questioning the accuracy of my diagnosis. You may well ask, therefore, why I widen the breach by introducing the last two cases, which, judged by the ordinary rules, belong in another category altogether, and which the writers mentioned do not appear to refer to at all. If, however, you recall the physical signs and anatomical features, you will recognize that they are well worthy of consideration in this connection. I have introduced them because they present some of the most striking features of capillary bronchitis. What else do the subcrepitant râles indicate here than elsewhere? Do they not show an affection of the smaller air-passages just as much as in the other cases? The difference is in the extent and course of the affection, not in the character. I have introduced them purposely, to show you that it is an error to apply the term capillary bronchitis to a physical sign in one case, and deny that it has the same significance in another of a similar character, when there is merely a difference in extent. One is as much capillary bronchitis as the other, and it ought to be recognized as such, whenever and wherever it occurs. Its connection with other physical signs, such as dullness, makes no difference if we have properly interpreted the subcrepitant râle itself. This view and the admission of the frequency of consolidation of the lung, from the extension of the disease beyond the bronchi, will explain all discrepancies, and make all cases clear.

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* 1866, p. 332.

But I have introduced the last two cases especially to show that the distinction usually made between capillary bronchitis and catarrhal pneumonia cannot be maintained, if we bear in mind the pathological process, and are not influenced by the idea that extent alone makes a difference in the nature of disease. In connection with this last point I will cite several authors, who not only confirm the view expressed about capillary bronchitis, but show how closely it is allied to catarrhal pneumonia.

The language of Jaccoud* is the most explicit:

"The physical signs vary according to the period and the lesions of the disease. At the beginning, when the bronchial obstruction has not caused any modification in the pulmonary parenchyma, percussion gives negative results. Later the resonance is slightly diminished on a level with collapsed portions of the lung, and is increased in front and at the apex, over the regions which are the seat of dilatation."

"The development of catarrhal pneumonia in the course of capillary bronchitis is announced by the elevation of temperature and by the appearance of pain with the cough. When the disease attacks only some isolated lobules, surrounded by the aerated lung, it does not modify the physical signs of the pre-existing bronchial catarrh, but if it is developed in the collapsed portions, which is generally the case, it ends by becoming confluent, and is revealed by dullness, increased vocal vibrations, the appearance of very fine subcrepitant râles," "by the bronchial character of the respiration, and by bronchophony. These last phenomena are caused by the condensation of the parenchyma by catarrhal infiltration. These physical signs are, in a word, those of fibrinous pneumonia. The difference is in the degree, they are less marked in catarrhal pneumonia, on account of the density of the infiltration being less; the dullness is less absolute as well as the loss of elasticity, and the vocal vibrations are less marked. The subcrepitant râles, however fine they may be, have not the characteristic fineness of the true crepitant râle, besides, in catarrhal pneumonia, the physical signs are often bilateral, and in doubtful cases the diagnosis may be facilitated by the consideration of the antecedents (previous capillary bronchitis) and by the termination of the disease; critical defervescence is the rule in fibrinous pneumonia, resolution by lysis in catarrhal pneumonia."

"The addition of the catarrhal pneumonia is grave, not only for the present but for the future, as persistent caseous infiltration is possible. The latter is particularly to be feared when pneumonia is limited to the apices. This last fact is exceptional, but what is less so, is to see a capillary bronchitis, with disseminated catarrhal pneumonia, end in resolution everywhere except at the apex. Though the immediate prognosis is favorable, the remote is grave, for such an individual will certainly show some day the symptoms of pulmonary phthisis."

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* *Pathologie interne*, Paris, 1872, vol. i., p. 789.

"The preceding explanation is, in every way, applicable to pseudo-membranous bronchitis." "The only differential sign is the expulsion of false membrane, flattened, tubular, or branched. Such expulsion is sometimes followed by recovery, but ordinarily the false membranes are reproduced."

We see in the above the clearest recognition possible of the frequent combination of changes in the pulmonary tissue with those of capillary bronchitis. The author even goes so far as to speak of capillary bronchitis and catarrhal pneumonia under the same head, and after stating that the inflammation of the larger air-passages genererally precedes that of the smaller, he follows the disease into the air-vesicles and finds not only catarrhal pneumonia, but in some cases croupous pneumonia, shows that the products of this inflammation of the pulmonary tissue may become caseous, and admits, as one of the results of the disease, even chronic catarrhal pneumonia. All these changes he speaks of as established facts without alluding to the views of others. The description, indeed, is such, that one who had acquired his knowledge from certain writers, and who believed that an absolute distinction was to be made between the various diseases mentioned, might find it very difficult to detect the boundaries.

We will now quote a late German authority, Lebert,[*] whose name inspires confidence, and whose description is admirable. He says: "We have repeatedly and strongly insisted upon the unity of pulmonary catarrh. We know that our views in this respect differ from those of most observers, and even competent ones, but from year to year we are more forced to insist upon this unity by the observation of transitional forms.

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[*] *Clinik der Brustkrankheiten*, vol. 1. p. 80. Tübingen, 1873.

"The signs of ordinary bronchitis generally precede those of capillary bronchitis. The latter may begin in the smaller air-passages, but it generally extends from the larger. But whether it be the initial disease or follow ordinary bronchitis, the attack is generally ushered in by fever, often rigors, and by dyspnœa. The rough, snoring, piping, and crepitant râles are mingled together. The sibilant sounds are fine owing to the swelling of the mucous membrane of the smaller bronchi. In slight cases we have with the ronchi vesicular respiration, but in the severer cases, the former cover up the latter. After the first few days, the sonorous râles are more rare, and the crepitant are limited to the inferior and posterior part of the lungs on both sides. Quite early one hears crepitus of medium fineness in inspiration and expiration. The character of this changes according to its exact position, whether in the medium, or very small bronchi, or in the air-vesicles. The subcrepitant râle, however, is most common in capillary bronchitis.

"There is no dullness, and the sound may be even more resonant than usual. In cases which occur in middle life a fatal termination is the more rare. Death generally takes place between the fifth and tenth day, but sometimes much later. Convalescence is slow and it may be some weeks before the signs disappear. The disease has also a great tendency to return."

Having thus spoken of capillary bronchitis, he describes, under a separate head, a broncho-pneumonic form of capillary bronchitis. "This is generally not primitive. One can generally, in a few days, observe the transitions from bronchitis to bronchiolitis and from this to broncho-pneumonia, or we may have intense bronchiolitis from the very beginning. When we have this combination, in addition to the subcrepitant râles, which are the unquestionable signs of bronchitis, and which have their maximum below and behind, we have dullness on percussion, then fine crepitus, and very soon bronchial respiration and bronchophony. The transitions between broncho-pneumonia and diffused pneumonia are seen all the more clearly the more carefully one observes, especially if both axillary regions are examined in every case. Every experienced clinician, especially he who has practiced much among children and the aged, will admit that, generally, broncho-pneumonia is much more inclined to catarrh, much more frequently arises from this, than it shows a relationship to diffused pneumonia."

These statements are strikingly in conformity with what our own cases teach. The combination of the various conditions is made very clear.

So many quotations have been made, I will do little more than refer to the description of catarrhal pneumonia by Juergensen.* On page 191 he says, "Acute catarrhal pneumonia originates in a severe catarrh of the finer bronchi, of great extent." On page 187 we are told that "catarrhal pneumonia is always a secondary process. It never begins in the alveoli. Usually it is preceded by an inflammation of the bronchial mucous membrane." There is much more to the same effect, but we will pass at once to Buhl,† whose views differ essentially from any of the above.

He first quotes Rokitansky as follows: "Not unfrequently the fine bronchi and extremities of the same are the seat of a very intense inflammation, which extends to the pulmonary texture. This appears in the form of numerous lobular points, dark-red, swollen, hepatized, or filled with pus."

Buhl himself considers catarrhal pneumonia an acute disease attacking particularly the lower lobes and extending from them. He objects, however, to the title, as there is no mucous membrane at the terminal extremities of the air-passages, which are covered with endothelium of lymphatic vessels. He considers that the process which takes place in the medium and finer bronchi, and which fills them with mucus and pus, and in which the alveolar parenchyma of the lung takes part, is no longer catarrh, in the usual sense of the word, and not even inflammation, for he thinks it very doubtful whether the muco-purulent contents of the alveoli are formed in them. He concludes by declaring that "what is called catarrhal pneumonia is only capillary bronchitis or bronchiolitis, in which the lung takes part by collateral œdema, atelectasis, local emphysema; and that the air-vesicles are filled in consequence of suction of the secretions of the bronchi into the lobules. He admits that pus corpuscles or colorless blood globules might come directly from the alveolar capillaries, but does not believe that mucus can be formed there, though he cannot absolutely deny such power, but its existence cannot be proved. He also admits that if the disease does not prove fatal, certain portions of the lung may never be restored, but remain in an emphysematous or collapsed condition, and, in the latter case, as air does not enter, the contained epithelium is transformed into a mass of pigment or caseous material. The mucus and pus may become immovable, degenerate, and obstruct as a caseous mass the bronchioles and lobules."

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* Ziemssen's *Handbuch der Speciellen Pathologie und Therapie*. Vol. 5. Leipzig. 1874.

† *Lungenentzundung, Tuberculose und Schwindsucht*. München, 1872.

These statements of Buhl are very interesting to us, as he admits the presence of the morbid products in the air-vesicles, while he denies that they are formed there. It is their presence which it is necessary to recognize, and we may leave for future decision the question of their exact origin.

If we recapitulate briefly the statements of the different authorities, we find all allow that capillary bronchitis is generally preceded by ordinary bronchitis. All, except Niemeyer, admit the gravity of the disease, and we may pardon him for treating it so cavalierly, as what is wanting here may be found under the head of catarrhal pneumonia, with which his name is so intimately associated.

In regard to the liability of the disease to extend to the air-vesicles, or pulmonary parenchyma, Valleix certainly does not call our attention to such a feature; Flint declares that the inflammation does not tend to extend beyond the smaller bronchi; Jaccoud admits the extension so fully as to blend the two diseases; Juergensen and Lebert describe such an extension in very exact terms; and Buhl speaks of the presence of the products of inflammation in the air vesicles, while he differs from the others in regard to their source.

In describing the physical signs all insist upon the subcrepitant râles, but they admit dullness and other signs of consolidation, just in proportion to the admission of the causes which produce them.

With such conflicting statements you may well ask,—what are we to believe?

After reporting, under the head of capillary bronchitis, cases which are described under different heads by different authors, the latter are quoted, and they run through all shades of opinion, some ignoring all changes, except such as are situated in the smaller bronchi, while others give a most prominent position to disease of the air-cells or parenchyma, until the circle is completed by a denial of any participation of the air-cells, except as a place of deposit for products formed in the bronchi.

How shall we reconcile these statements? Simply by describing things as they are. For many years I have been looking for such cases as are usually described under the head of capillary bronchitis, for pure examples of the disease, until the conviction has been forced upon me that such are very rare, if the whole chest be carefully examined, and no observations in which any part of the chest is overlooked can be accepted as complete. Having reached the conclusion myself, that cases of the kind are generally mixed, I hoped to lighten your labors by giving you the result of my experience. To make the points clearer, it was thought best to quote the opinions of certain authors, whose views appeared to require some modification. But, on examining more closely the literature of the subject, I found most unexpected support.

The description given by Lebert is very accurate, but even this needs some explanation. It was not written with reference to the doubts which might arise in the minds of those whose views were obtained from other sources, and who were endeavoring to get an accurate idea of a disease of which experience had as yet taught them nothing.

Let us, therefore, guided by the light of our own experience, and that of others, endeavor to give such a description of the disease as will make all varieties of cases clear that are really allied, and prepare us to recognize the affection under any of its forms.

Ordinary bronchitis, or inflammation of the larger air-passages, in the vast majority of cases, is a slight affection and soon disappears.

Sometimes, but very rarely, the inflammation extends more deeply, and attacks the smaller air-passages. When this occurs, or when the latter are attacked primarily and a large portion of them are involved, there are fever, marked dyspnœa, urgent cough, with muco-purulent expectoration. An examination of the chest shows resonance on percussion, when the bronchi are alone affected, and sibilant, or more frequently subcrepitant râles, the latter being the characteristic sign. These cases are much more serious, and often terminate fatally. To these the term capillary bronchitis has been applied. In most descriptions given we are told that this affection is always bilateral and extensive; but there is a class of cases in which the physical signs are absolutely the same; the subcrepitant râles differ in no way; they are only confined to a limited portion of the chest. They indicate the existence of capillary bronchitis as much as when the bronchioles of both lungs are affected. Though they may soon disappear, their import is always grave, unless they follow acute declining pneumonia.

They are the signal of danger either immediate, from their extent, or remote from the sequelæ, whether universal or confined to a very limited spot, and this danger should be recognized and acted upon.

When the above symptoms alone are present, the term capillary bronchitis is certainly applicable, but the frequency with which the inflammation involves the air-vesicles, and gradually causes consolidation of the lung, is not generally stated with sufficient distinctness. So much stress has been laid upon resonance that dullness is supposed to exclude the disease, while it is only the evidence of a very common accompaniment, both in cases where the affection is general, and in those where it occupies but a limited portion of lung. The changes which these consolidated portions of lung undergo may be traced until they end in the destruction of the pulmonary tissue, and the process may be repeated in one portion of the lung after another. The subcrepitant râles of capillary bronchitis may be mingled with the fine dry crepitant râles, which indicate an affection of the air-cells, and which differ in no way from those heard in ordinary croupous pneumonia. In some cases, indeed, the mode of attack and the physical signs resemble in all respects those of ordinary pneumonia, but resolution does not take place, the dullness and subcrepitant râles persist, and the patient finally sinks with the diseased portions of lung partly destroyed, partly in a state of caseous degeneration.

To this last class of cases, in which the pulmonary tissue is involved, the term catarrhal pneumonia has been applied, but they certainly have much in common with capillary bronchitis, if we bear in mind the anatomical lesions, and compare them with the third and fifth cases reported. In the first of these the bronchi were principally and most extensively affected, but the microscope showed also a great variety of changes in the air-vesicles, some of which were certainly such as are described in connection with catarrhal pneumonia, which name would certainly be applicable to the fifth case. Juergensen, it is true, states that catarrhal pneumonia never begins in the air-vesicles. It is difficult to understand how one can make so positive a statement, unless it be allowed at the outset that the term catarrhal pneumonia shall only be applied to a form of disease which begins in the air-passages. But, whatever idea the author intends to convey, you will probably meet with cases, in which, though seen early, the first physical sign detected will be the fine dry crepitant râle which has its seat in the air-vesicles, and after a time you will hear the subcrepitant râle of the smaller air-passages. It would be impossible to show that the inflammation did not begin in the air-passages, but the evidence of such a beginning is wanting.

But, by whatever name you call the disease, you may be sure of its nature, if you use proper care in acquiring a knowledge of the physical signs. If you interpret the latter properly you cannot be mistaken. If fine, dry râles indicate that the air-vesicles are affected, accept the fact. If subcrepitant râles are heard in any part of the lung, they indicate more or less fluid material in the smaller air-passages. The history of the case and the other signs will enable you to decide whether the material is attributable to inflammation, and, if so, you have capillary bronchitis, and if these physical signs extend over the greater part of both lungs you have the capillary bronchitis which authors have intended to describe. The detection of the morbid process as such is the first point. The fact that it is combined with other changes should not lead us to ignore it, however limited it may be. Neither should we fail to recognize the changes which accompany it; we must give them their due weight. Bearing in mind the significance of dullness, and having satisfied ourselves that it is connected with some disease of the pulmonary tissue itself, we must decide, if possible, whether it be caused by collapse, œdema, or by the accumulation of morbid products in the air-vesicles. But we will not fail to give due credit to those who have made so clear to us the striking characteristics of certain forms of the disease. Though the outlines are sharper than a survey of the whole subject will warrant, the different parts of the picture are in the main correct. Our object is not so much to reconcile the conflicting statements of authors as to blend and arrange them in the proper order, and give them due proportion.

We see here an example of what has occurred in connection with the investigation of other diseases. First, differences are noticed, and the tendency is to make such differences prominent. Then experience and closer observation show the connection between these apparently different things. Before the time of Hebra most laborious and faithful descriptions were given of affections of the skin supposed to be of a different character, but he grouped them all under the head of eczema, regarding them as only transitional forms of the same process manifesting itself in different degrees and in different ways, some of which are much more common than others. It is the very accuracy of those who have made such distinctions that has enabled us to see how these processes blend with each other.

Having an accurate idea of the anatomical changes which take place in the diseases we have been discussing, we are prepared to understand the variety of names which have been applied to them, such as catarrhus suffocativus, capillary bronchitis, bronchite capillaire, purulente and pseudomembraneuse, bronchitis diffusa, bronchiolitis, broncho-pneumonia, catarrhal pneumonia.

The above views will enable you to understand, clinically, all th varieties alluded to, while you may leave for more accurate ob servations all details of the exact condition of the diseased lobules of the lung.

Whether you regard the lining membrane of the air-vesicles as endothelium or epithelium ; whether the morbid contents of the air-cells are sucked in from the bronchi, are the product of proliferation, or are derived from blood-vessels, through whose walls they have passed; whether they are composed mostly of fibrin or cells; whether you have the microscopical characters of croupous pneumonia, or catarrhal pneumonia, or desquamative pneumonia, or interstitial pneumonia, or peribronchitis, you may know that there is more or less consolidation of the lung. Further researches, a more accurate comparison of the symptoms with the microscopic results are required before you can pronounce with certainty upon these nicer points. But you may know the relative amounts of bronchial and parenchymatous disease with sufficient accuracy for clinical diagnosis. At some future time you may be able to describe the exact character of the contents of the air vesicles while standing over the living subject of disease; you can do it only approximately now.

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THE INFLAMMATORY ORIGIN OF PHTHISIS.

BY

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The patient whom I have brought before you this morning is suffering from phthisis, a disease which is certainly common enough to justify us in holding very decided views in relation to its pathology. And yet there is perhaps none concerning which a greater revolution in sentiment has taken place since I quitted my place on the benches you now occupy, less than a score of years ago. At that time the gentlemen who occupied the positions of clinical lecturers here, taught with Laennec, that phthisis was in every case dependent upon a new or morbid formation in the lungs, which softening in time and being expectorated, gave rise to cavities. This was the prevailing theory until within a very few years, when the important part which inflammation plays in the production of this disease was recalled to the minds of thinking physicians. I say recalled, because Niemeyer was by no means the first who held that the lesions of phthisis could almost invariably be traced back to inflammation, and that this process was primary and not secondary, as taught by Laennec and his followers. I have no time, and, indeed, this is not the place to go fully into the literature of this subject, and I must therefore content myself with a very few references, which will be, however, sufficient to prove the correctness of my position.

Hippocrates* referred consumption to suppuration of the lungs, which might be due to pneumonia, hemorrhage, or an accumulation of secretion. But the ancients appear to have had no idea of the nature of what is now called tubercle. To pass to more recent times, it would be easy to show that during the past forty years there have been several writers who have put on record their disapproval of Laennec's theory. In 1845, Dr. Thomas Addison,† in an address delivered before the Guy's Physical Society, expressed the opinion that "inflammation constitutes the great instrument of destruction in every form of phthisis." No one can, I think, read this admirable paper without coming to the conclusion that its accomplished author has anticipated Niemeyer in almost every point of importance. Again, in a work entitled "Outlines of Pathology and Practice of Medicine," which was published in 1844, Dr. William Pulteney Alison says, when speaking of the terminations of inflammation, "From these facts‡ it seems reasonable to infer, that in certain constitutions tubercles and all their consequences are direct effects of inflammatory action, and may be prevented if that action be arrested or subdued." In another place the following very similar language is used: "The deposition of tubercles§ may be, and often is, the result of an action to which it would be absurd to give any other name than inflammation." Very similar views appear to have been entertained at about the same time by Dr. C. J. B. Williams. Dr. Alison, it is true, does not distinguish between the two conditions now known as tuberculosis and phthisis, but the sentences I have quoted from his work show not only that his views in regard to the pathology of the disease were sound, but that he was also well informed as to its proper treatment.

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* Quoted by Ruehle. Ziemssen's "*Handbuch der Speciellen Pathologie und Therapie.*" Bd. V.

† "A Collection of the Published Writings of the late Thomas Addison, M.D." The New Sydenham Society. 1868. P. 64.

‡ Page 199. § Page 196.

It is strange that papers so complete as those I have referred to should have exercised so little influence, and that Laennec's opinions should have, in spite of them, been so universally accepted. You are all doubtless fully aware how much the writings of Niemeyer have contributed to popularize a theory which was so nearly forgotten, but it would be unfair to attribute the change that has taken place wholly and solely to his influence, great as it has been, for he has been sustained in his opinion by numerous other German pathologists, and among them by Buhl, Waldenberg and Rindfleisch. The last-named, it is true, seems lately to have somewhat modified * the opinions he formerly held as to the connection between phthisis and tuberculosis, but he does not deny the dependence of the former upon inflammation.

Without further prelude, I will read the notes of the case before you, directing your attention afterwards to some points in the pathology and treatment of phthisis, which it seems to me are specially illustrated by it.

G. B., æt. 35, a sailor, and a native of Maine, was admitted into the men's medical ward of the Pennsylvania Hospital, January 8, 1875. His habits have been very intemperate for several years past, but with the exception of a severe attack of facial erysipelas which occurred about two years ago, and from which he made a good recovery, he has had perfectly good health up to the beginning of his present sickness. He is also free, so far as can be ascertained, from hereditary predisposition to consumption or any other form of disease.

Last August, in consequence of imprudently exposing himself to the cold air when overheated, he was taken with a feeling of oppression in the chest and of general prostration. Cough does not appear to have been at this time a prominent symptom ; he says, however, that he was obliged constantly to clear his throat. He took very little care of himself during this attack, never at any

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* Ziemssen's "*Handbuch der Speciellen Pathologie und Therapie.*" Bd. V.

time, he tells us, taking to his bed, although feeling wretched and weak. In September, he had several trifling hemorrhages, and one which was profuse, in consequence of which he was admitted here on the 29th of the same month. At this time the physical signs indicated the existence of phthisis, but so great an improvement in his condition took place, that he asked for his discharge on October 12. He then returned to his occupation, which he was able to follow until November 10th, when, after having been thoroughly wet during a storm, he had a recurrence of the feeling of oppression, accompanied by a sharp pain in the left side of the chest. He says he has lost flesh, but has never suffered to any extent from night-sweats. An examination of his urine yielded only negative results. He was readmitted to the hospital on January 8, when the diagnosis which had been previously made, was confirmed by the physician then on duty. The treatment adopted consisted in the administration of cod-liver oil, together with the syrup of the iodide of iron. In consequence of the recurrence of the hemorrhages, the latter was replaced by the solution of the subsulphate.

On the first of February, the patient was transferred to my care. His general condition at that time was good; he had little or no cough, not much expectoration, no night-sweats, and no spitting of blood. He ate and slept fairly. The physical signs, briefly stated, were as follows: No depression under either clavicle, feeble expansion of left infra-clavicular region. Vocal fremitus more marked over left infra-clavicular region than over right. Dullness under left clavicle, extending to fourth rib, except over the second interspace, two inches from the sternum, where a cracked-pot sound was obtained. Posteriorly, dullness over the left superior scapular region. Beneath the left clavicle numerous crackling sounds were heard, and in the second interspace, cavernous respiration and pectoriloquy. Posteriorly crackling was heard in the superior scapular region of the left side. On the right side a few indistinct râles were heard in front, but only after coughing. The physical signs revealed nothing abnormal at the lower part of the chest, either in front or behind. A loud systolic murmur, harsh in quality, was heard at the base of the heart, to the left of the sternum. The patient has fistula in ano. He is reported to have had a convulsive seizure on the evening of January 30th, for the first time in his life. It seems to have been epileptiform in character.

February 9. The patient had vertigo this morning, accompanied by a "bad feeling" in his head, but unaccompanied by loss of consciousness. No change in the fundus of either eye can be detected with the ophthalmoscope.

Feb. 26. An attack of facial erysipelas of moderate severity came on yesterday, for which twenty drops of the tincture of the chloride of iron every three hours were prescribed, together with six grains of quinine daily.

Feb. 29. Erysipelas subsiding. In consequence of the existence of great prostration, six ounces of whisky were added to the other remedies.

March 4. A slight hæmoptysis occurred this morning. The patient was ordered fluid extract of ergot (fzss. t-d). Other treatment suspended. A ligature has been passed through the fistula, so far without much benefit to the patient.

I will omit the further notes of this case, and pass on at once to speak of the patient's present condition. This is good. Since the date of the last note, he has had one or two slight hemorrhages. He is very little troubled with cough, in spite of the persistence of the physical signs indicating disease of the apex of the left lung. It is not necessary to recapitulate them, since they do not differ materially from those noted as present on February 1st. There is, therefore, no reason for thinking that the disease has extended; on the contrary, I believe the reverse is the case, for the number of moist râles have very decidedly diminished. The pulse, too, is scarcely accelerated, and has not been so except during the attack of erysipelas, or just after a hemorrhage. The temperature, too, has never been much elevated, except at the times just referred to.

Let me now recapitulate briefly what seems to me to be the important features of this case. Our patient, who has not yet reached middle life, notwithstanding an entire freedom from hereditary tendency to phthisis, now presents physical signs which indicate with great certainty the existence of this disease, and this, I may add, is not only my own opinion, but that of two of my colleagues. Unquestionably his naturally strong and robust constitution has been gradually broken down by his intemperate habits, and the recklessness with which he has exposed himself when intoxicated, since there is no evidence of the existence of lung disease prior to the attack of sickness in August. But in the month following, the changes in the lung were sufficiently marked to enable my colleague, Dr. J. Forsyth Meigs, to make a positive diagnosis. A slight improvement in our patient's condition then rapidly took place, and induced him to ask for his discharge. But he had not learned wisdom from experience, and as a consequence of imprudence in exposing himself, had a second attack of illness similar in all respects to the first, but more severe. This led to still further lesions of the lung. There appears to have been but little cough, and but little expectoration during these attacks, from which I infer that they were not complicated to any extent by bronchitis; it being a well-known fact, that the cough which occurs in pneumonia owes its existence much more to the intercurrent bronchitis than to the inflammation of the lung itself. Our patient says that, although suffering on these two occasions from oppression and fever, he did not have to give up his work at any time, which renders it improbable that he suffered on either occasion from the form of pneumonia which it is now the fashion to call croupous, to distinguish it from another variety which is designated as catarrhal.

With the exception of a few moist râles in the right superior scapular region, which were only heard after the act of coughing, and which have almost wholly disappeared, leaving behind them no sign of softening, the evidences of disease of the lungs are now confined to the upper part of the left lung. From this fact, as well as from the absence of hereditary predisposition, I regard the case as one in which the disease is of local origin, and traceable to the pre-existing inflammatory process. It is rare, and it is fortunate it is so, that in the absence of constitutional tendency this result occurs, but you must remember that a predisposition may be acquired as well as inherited, and that nothing tends in a greater degree to bring this about than habits of debauchery and the neglect of health which these generally engender. Another feature of interest in this case is the constantly recurring hemorrhage.

I have given it as my opinion, that the patient has had at least two attacks of catarrhal pneumonia. The existence of this form of pneumonia has been called in question by many accomplished pathologists, but my experience leads me to range myself with those who contend that there is a variety, differing entirely from the sthenic form in the lesions it produces, in its mode of approach, in the degree of febrile reaction by which it is accompanied, in its physical signs, and in fact in almost every clinical aspect. It differs also in the kinds of individuals it attacks. Sthenic, or croupous pneumonia, is most common in adults or adolescents who were in good health at the time of their seizure. Catarrhal pneumonia is to be found more frequently among the very old or the very young, or in weakly people who have been debilitated by previous disease or other cause. It does not usually begin abruptly, as is the case with the other form, but it is generally preceded by bronchitis, and its onset is frequently so unmarked by special symptoms that if the possibility of its occurrence be not borne in mind, it may be, and probably is, often overlooked. The former indicates, therefore, a greater vigor of constitution than does the latter. The former invades a whole lobe of the lung, the lower lobe being its favorite seat, while in the latter the inflammatory process, extending insidiously from the bronchial tubes, more often affects scattered lobules, those situated at the apices being most liable to be invaded. The physical signs by which the two forms are recognized, are also different. You will not find in the latter as marked bronchial respiration, bronchophony, dullness or vocal fremitus as in the former. Nor do we have the crepitant râle in as great perfection, in fact, it is often absent, and its place is taken by subcrepitant râles which owe their existence to the accompanying bronchitis. The rusty, viscid sputa, pathognomic of the former, is never present in the latter, which gives rise to an expectoration catarrhal in character, but which, under the microscope, is found to contain in great abundance the cells lining the alveolar walls. Care will, however, always enable us to recognize the presence of catarrhal pneumonia, for it is always accompanied by a marked diminution in resonance over the parts of the lung affected by it. Moreover, it may be known to have occurred in the course of bronchitis, when, in conjunction with other symptoms indicating its presence, a decided alteration in the pulse ratio takes place, *i.e.*, when the respiration becomes relatively frequent so as to bring about some approach to the condition which is usual in the sthenic form of pneumonia. It will be rare, too, if under these circumstances careful thermometric observations do not detect the existence of an exacerbation of fever.

The principal objection made to the admission of catarrhal pneumonia is that the mucous membrane of the bronchial tubes does not extend into the air-cells, that these have no epithelial lining, and that consequently no process beginning in the former can extend into the latter. The truth of the first portion of this objection must be admitted, but modern research has demonstrated the existence of a layer of cells lining the alveolar walls, but having the characters of an endothelium rather than of an epithelium.

The resemblance of these cells to those of lymphatic tissues is very striking. In fact some histologists have gone so far as to contend that the lymphatic vessels in the lungs communicate with these cells. These facts enable us to explain the existence of tubercles in the alveoli; most microscopists looking upon tubercles—and I am now, of course, making a distinction between phthisis and tuberculosis—as minute lymphadenomata.

Admitting therefore, as indeed it seems to me we must, the existence of this endothelial layer, there is no reason why the cells which compose it should not, under the stimulus of inflammation, occasionally undergo proliferation. In very young children the inflammation of the alveoli does not invariably arise by extension from the bronchi, but takes its origin sometimes in the following way: A bronchial tube becoming clogged with secretions, tough and viscid in character, no longer conveys air to the cells which depend upon it. But this plug of mucus, acting as a valve, while it prevents the entrance of air, permits its escape. Under these circumstances different lobules will soon be exhausted of their supply, and collapse will ensue, a condition on which inflammation is exceedingly likely to follow. In adults, collapse is less common, but it should be remembered that it may occasionally happen. More common it is for the retained secretion to undergo decomposition, and hence to become an irritant.

I have told you it was the lower lobe which was the most frequently affected in the croupous form of pneumonia, and that it was the upper in the form under consideration, as in the case before you. Let us see whether we can find a satisfactory reason for this. A little consideration will convince you that the apices are the parts most liable to be invaded by an inflammation extending from the bronchi. These parts of the lungs are, even in health, more quiescent than any other, and hence it is in them that accumulations of mucus or of epithelial débris will be most likely to take place whenever the respiration is feeble. Moreover they are not so thoroughly emptied of these accumulations by the act of coughing. Indeed it is perfectly possible that while this may dislodge the secretions from the lower lobes it may drive them to the upper, and for the following reason. The upper portions of the chest-walls are provided with less powerful muscles of expiration than the lower, and consequently during the effort of coughing, which is nothing more than a forcible act of expiration performed with the glottis closed, the air and secretions may be driven from the lower portions of the lungs into the upper. Hence it is that we see in emphysematous lungs the lesions most marked at their apices and along their free borders. In an enfeebled condition, such as usually precedes phthisis, this disparity in power becomes still more marked ; in fact we may have what has been called by the Germans, the paralytic thorax. Now this condition is unquestionably congenital in many instances ; it may be acquired, however, and much may be done by the use of proper hygienic measures, not only in preventing it, but also in moderating the ills which flow from it. Let us suppose the air-cells inflamed in consequence of the impaction of epithelial débris, we can then very readily understand that the further tendency of the disease will be to a congestion of the pulmonary vessels of the part, the swelling of the tissues, and the proliferation of the endothelial lining of the air-cells—a condition giving rise to physical signs identical with those formerly attributed to the stage of deposition, for we have dullness on percussion, diminished movements of the affected side, increased vocal fremitus and resonance, and broncho-vesicular respiration. Now, it is perfectly possible that from this condition the patient may make a good recovery ; the contents of the alveoli undergoing fatty degeneration and being absorbed. But this fortunate result is one which experience will not allow us to look for frequently in scrofulous subjects ; for those who have no hereditary tendency to phthisis there is more ground for hope. In the former class it is much more usual for further changes to manifest themselves. But these may not occur immediately, and the length of time which elapses before they do, will, to a certain extent at least, determine the nature of those changes. If but a short time intervenes we shall have a rapid caseous degeneration of the products of the inflammation taking place, together with a loss of vitality of the part in consequence of the cutting off of the supply of blood, either in whole or in part, by the pressure of the accumulated cells within the alveoli. Softening now supervenes, which is, in its turn, followed by the expectoration of the caseous masses and broken-down tissues, and as a consequence, by the formation of cavities.

If, however, the resistance made by the constitution is greater, the patient may regain his health ; gradual disintegration and absorption of the caseous masses taking place. In these cases, instead of the breaking down of the lung, an increased development of the interstitial connective tissue is found to have occurred. It is rare, except in very acute cases of phthisis, not to find these two processes commingled to a greater or less extent. The more rapid the progress of the disease, the more likely will we be to find caseous degeneration only ; while, on the other hand, in chronic cases, there is a decided tendency to the development of what is known as fibroid phthisis. The existence, therefore, of an increased growth of connective tissue, and the part it plays, should not be forgotten. In fact many of the physical signs of phthisis are more correctly attributed to it than to the caseous degeneration. Among these may be mentioned the falling in of the infra-clavicular spaces, and the displacement of the heart and trachea. Very frequently the contraction of this tissue causes the enlargement of existing cavities, and not rarely has it brought about so much enlargement of the bronchi, that these dilatations have been mistaken, both during life and after death, for vomicæ. There is another way in which this increased development of connective tissue may exercise an important influence upon the further course of the disease. But I speak with much hesitation on this point, as it cannot be said to be proved. It may encapsulate the caseous masses so as to prevent their absorption, and the consequent infection of the blood, which gives rise to tuberculosis. It has always been a difficult thing for the partisans of the theory of infection to explain why an eruption of tubercles occurs so early in one case, while it is so long delayed, or may never appear, in another. It seems to me that this development of connective tissues may, in some cases at least, explain this fact.

In the patient before you, the physical signs indicate, with great distinctness, the existence of softening, which has advanced so far in the left lung as to form a cavity in the upper lobe at almost its middle. There is no question in my mind that this process is the predominant one. The other may exist, but it is certain that it holds a subordinate position.

I may here very properly allude to the views taught by Buhl. This distinguished pathologist, the author of the infection theory of tuberculosis, while he holds that inflammation is the active element in the production of phthisis, believes that it is seated in a different part of the lung from that in which it is placed by Niemeyer and others. Rejecting the term catarrhal pneumonia as improper, because there is no proper mucous lining of the air-cells. he calls the form of inflammation of the lung which most frequently precedes phthisis, acute desquamative pneumonia, holding that the interstitial tissue is the true seat of the inflammatory process, and that the increased proliferation of cells is but the effect of an irritation propagated to the walls of the alveoli. The desquamation is therefore a consequence and not the essential phenomenon of the disease. The difference between this view and that which I have adopted is, therefore, rather in regard to the origin of the process than as to its results. The supporters of the latter view believe that the inflammation is primarily superficial, involving later the more deeply-seated structures, which in the end lose their vitality in consequence of the pressure exerted upon them by the cells accumulated within the alveoli. The upholders of the other view maintain the reverse, teaching that the same result is brought about by the effusion in the tissues of the lungs themselves. They both hold in common that as an effect of the inflammation we may have caseous degeneration, and a growth of connective tissues.

It may occur to some of you that I have misinterpreted the patient's symptoms, and that he is now suffering from the effects of an ordinary attack of acute pneumonia of the apex, which, instead of undergoing resolution, has given rise to an abscess. The cavity, if this explanation be correct, would then be the result of acute suppuration, rather than of the gradual breaking down and throwing off of tissue. I have considered, in all its aspects, this interpretation of the case, and I cannot find that it rests on an at all substantial foundation. In the first place the history of the case affords no support for this view ; for it tells us that the patient has never, during the whole course of his illness, had a marked chill, rusty, viscid sputa, or a sudden expectoration of pus, as he would have had from the rapid emptying of an abscess. Moreover, he has not been ill enough to have had an attack of acute croupous pneumonia ; on the contrary, expectoration has been scanty, and the symptoms which he has constantly presented could only have been due to a slow, insidious disease. The slight extent to which he has been annoyed by cough is certainly unusual, but it is not so much so as to prove our diagnosis incorrect. It is also not inexplicable, for it is well known that the frequency of the cough, and the annoyance arising from it, are in no degree proportionate to the extent of lung invaded by phthisis, but rather to the severity of the bronchitis which accompanies it. I have had other cases under my care in which, notwithstanding the presence of pneumonia, cough was of infrequent occurrence. A few weeks ago a patient was admitted into the medical wards with consolidation of the apex of the left lung, which was just beginning to break down. It was impossible to obtain from this man an admission that cough had been at all a prominent, or even an annoying symptom. In fact so frequently is it absent that I am convinced that pneumonia often runs its course unsuspected by the practitioner, whose attention is directed to the chest only where it is present. The case I have referred to is by no means an isolated one in my experience, and I am therefore convinced that pneumonia of the apex is frequently overlooked, because the possibility of its existence is not sufficiently often taken into consideration. In a case of phthisis which I have had under observation for several years, the disease began in pneumonia, which, in consequence of its not presenting many of its usual symptoms, very narrowly escaped detection.

An interesting feature in the case before you is the frequency with which hemorrhage has occurred. It is probably well known to most of you, that Niemeyer holds that a hemorrhage is often the starting-point of consumption, the blood retained in the air-cells undergoing decomposition, and thus proving a source of irritation ; this has been denied by other observers, who contend that the hemorrhage is an indication of changes in the lung which have been overlooked. This is a difficult point to decide in a way that shall be satisfactory to all. To me, there seems to be nothing unreasonable in the idea that in a debilitated condition, a bleeding may occur in consequence simply of a loss of nutrition on the part of the vessels of the lungs. In fact, we have abundant evidence that it takes place frequently in individuals who neither are at the time nor subsequently become phthisical, and in whom no lesions of the lungs can be discovered after death, and it is therefore possible that it may sometimes be the first overt act, if I may be allowed the expression, of a phthisical predisposition.

There seems to me, however, to be abundant evidence, both clinical and experimental, in favor of Niemeyer's view. Dr. Julius Sommerbrodt* has shown that the injection of blood into the lungs of dogs, gives rise to lesions closely resembling those usually ascribed to catarrhal pneumonia, and there is therefore no great stretch of the imagination required to believe that the blood which is retained in the air-cells after hemorrhage, will give rise to precisely similar results in man. Having catarrhal pneumonia, and in many cases an hereditary tendency to consumption, the further changes will not be slow in appearing. The clinical evidence in favor of this view also appears to me to be conclusive. During the last few years, with the view of reaching a correct conclusion on this point, I have kept careful records of a number of cases in which hemorrhage occurred while they were under my care. In all of these, a day or two after its occurrence, an increase of temperature was observed, and this exacerbation took place no matter how frequent the recurrence of the hemorrhage. Later, the physical signs indicated an extension of the disease. I can well remember the case of a woman about middle age, who was under my care three years ago, and who was the subject of repeated hæmoptysis. During the intervals the temperature scarcely rose above the normal point, but two days after a hemorrhage, a rise with evening exacerbations was invariably observed. Dr. C. J. B. Williams, who opposes the theory that a hemorrhage may occasionally be the starting-point of phthisis, cites in support of his opinion the familiar circumstance of the retention of blood in the retro-uterine sac for indefinite periods without its giving rise to peritonitis. But he has overlooked this important point, that the air does not in that case gain access to the blood, and that, hence, decomposition does not take place.

Inasmuch as there are still many who doubt the inflammatory origin of phthisis, I have thought it might be to your advantage to collect the notes of the cases of this disease which have been under my care in the wards of this hospital since the beginning of my connection with it in 1868. During the seven years that have elapsed since then, I have treated seventy-three cases of phthisis, of all of which I have preserved notes. These have been taken by different gentlemen, and are not always quite so full in regard to the manner in which the disease originated as I could wish, but they are so, fortunately, in a sufficient number of instances for our purpose. In fifty-three cases, the patients were able to trace their illness back to a definite commencement, and in many of these cases (31) this appears to have followed directly upon a thorough wetting or chilling. Allowing for all sources of error, and especially for the proneness of patients to deceive themselves in all matters connected with this disease, it does seem to me impossible to draw any other conclusion from these figures, than that phthisis must at least sometimes be a sequel to inflammation of the lungs. In fact, every practitioner of medicine of large experience must have met with patients in whom he has been able to follow out every step of the disease from its beginning in bronchitis or catarrhal pneumonia to its ending in phthisis.

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* Virchow's *Archives*, June, 1872.

Another proof, if any more be needed, that consumption is a disease of inflammatory origin, is its occurrence as a frequent sequela to many constitutional diseases in which bronchitis is a constant complication. I need not recall to your memories how frequently phthisis follows measles, whooping-cough, typhoid fever —all diseases having a tendency to the production of bronchitis. This is a fact known as well to the laity as to the members of our profession, although the connection between these diseases and consumption has only recently been clearly made out. The popular saying, therefore, that consumption is the consequence of a neglected cold, is more nearly correct than is admitted by Laennec and his followers. It is certainly one which we can no longer afford to sneer at; on the contrary, it is, I believe, sound pathologically, and equally so are the indications it affords for treatment. It cannot be objected to this, that consumption has no further connection with these diseases than as a consequence of the debility left by them ; for diseases of equal severity and duration, but without tendency to pulmonary complications, are certainly much more rarely followed by consumption. Among the latter may be mentioned scarlet fever, small-pox, and typhus.

Remember, therefore, that a cold, or to speak more properly, a bronchitis, it matters not how slight and unimportant it may appear, is never to be lightly thought of in a person predisposed to phthisis, and remember, also, that the predisposition may be acquired as well as inherited, and that no matter how strong and robust the constitution may originally be, if prudence be cast aside and all the rules of hygiene be violated, if a course of unrestrained debauchery be entered upon, and if as soon as one cold is cured another be contracted, any one may at length fall a victim to consumption, as has the patient now before you. He has this advantage, however, over one with hereditary tendencies to the disease, that in his case the prognosis is more favorable. For, while part of the damage done to his lungs is irreparable, there is every hope that we shall succeed in preventing the extension of the disease. This hope is strengthened by the marked improvement which has already taken place. His prospects of recovery would be very much increased by an abandonment of his previous habits, since a return to these would almost inevitably be followed by a recrudescence of the morbid process.

It has been objected, rather foolishly I think, to this theory of phthisis, that it is calculated to mislead in practice, by causing the adoption of a line of treatment different from that now in vogue, and to the consequent employment of antiphlogistic remedies. I need scarcely say to you that there is no force in this objection. I hold that if the theory be true it should be taught, believing that the cause of science is never injured by the promulgation of the truth. But I cannot conceive what ground there is for fear that this will lead us into error, for it is no longer the fashion to treat even sthenic inflammation by debilitating remedies. On the contrary, the results of experience show that the best results are obtained in croupous pneumonia by the exhibition of tonics and nutrients. It is, therefore, extremely improbable that depressants would be employed in the management of an inflammation, the typhoid tendencies of which must be apparent to the most superficial observer. In point of fact, depressing remedies are not so used, physicians who have become converts to the theory of phthisis which I have endeavored in this lecture to explain to you being prominent among those who condemn their employment. Moreover, a knowledge of the pathology of the disease enables us to guard our patients against exposure to causes which may either aggravate an existing consumption, or induce it where it has not yet appeared.

I have said little in regard to the relation which phthisis bears to tuberculosis, beyond alluding to the fact that the latter is regarded by the majority of the pathologists of the present day as in some way dependent upon the former. According to the Germans, it is an *infektion krankheit*, the infection being brought about by the absorption of the caseous masses resulting from the products of catarrhal pneumonia. As I have already told you, it is difficult to explain why, in some cases the deposition of tubercles takes place so soon after the beginning of a phthisis, and why in others it is so long delayed ; but the co-existence of these two conditions is so frequent, that it seems unreasonable to deny all connection, more especially since this connection appears to be proved by the results of inoculating some of the lower animals with caseous matter derived from the lungs of consumptives. Rindfleish * has recently modified his views on this point, teaching now that the changes in the air-cells in phthisis are essentially tubercular, but that they are always preceded by bronchitis, the products of which in scrofulous subjects are capable of producing the infection. To go further into this matter would require more time than is at present at my disposal, and I must therefore pass on to speak of the treatment which has been adopted in the case before you, and that which is applicable to the disease generally.

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* Ziemssen's "*Handbuch Fünfter Band.*"

If there is any truth in the theory I have been endeavoring to inculcate, that phthisis is usually a disease of inflammatory origin, the chief indication for its treatment will be to attempt to set limits to the inflammation, and to undo, so far as possible, its effects. In fact, before the actual outbreak of the disease, we should always have in mind the great importance of protecting those predisposed to it against exposure to the causes of pneumonia. In this way we may often succeed in averting it entirely. But there is another indication which is equally important, and that is, to maintain the strength of your patient. We are not, therefore, to keep those with a predisposition to phthisis, or even those in the early stages of the disease, too closely confined to the house, for by so doing their health is undermined, and they are rendered more susceptible to the causes of inflammation. On the contrary, experience has shown that the best results are obtained by allowing our patients to live in the open air as much as possible, and by recommending them to avoid all in-door employments. They should be directed, however, to protect themselves, by suitable clothing, against cold and wet. In the treatment of catarrhal pneumonia, if it is necessary to prescribe antiphlogistic remedies, we should use those which, while they have the power to reduce the temperature, have no tendency to depress the strength. I have, therefore, been in the habit of administering in this condition, no matter whether it occurs as an original disease, or whether it is an intercurrent attack in the course of phthisis, from six to eight grains of quinine daily, either alone or in combination with opium and digitalis, in the form of what it is usual to call Niemeyer's pill. If the bronchitis accompanying the disease be severe, I have found the muriate and carbonate of ammonia, in doses of from five to ten grains, of great service in promoting secretion and expectoration. If the cough be dependent simply upon irritation, a teaspoonful of the syrup of wild cherry, with a few drops of the deodorized tincture of opium and of sulphuric acid is a more useful prescription.

In the intervals between the attacks of inflammation—and there will be many such in the course of chronic phthisis, when the patient is entirely free from fever, and may delude himself, and perhaps others, into a false feeling of security—our remedies should be tonic in their properties. In this class none will be found more generally serviceable than cod-liver oil, which should be given as long as it is borne by the stomach. Its use should be abandoned, however, whenever it provokes vomiting, or even when it creates nausea, as it is of the greatest importance that neither the appetite, nor the powers of digestion, should be in any way impaired. I generally intermit the administration of the oil during attacks of fever, as I have found it more likely then than at other times to disagree with the patient.

It is impossible within the limits of a single lecture to lay down a plan of treatment which shall be applicable to every case. Phthisis, in spite of a general impression to the contrary, is a disease capable of presenting itself under various forms, and he will be most successful in its treatment who is aware of this fact, and is prepared to meet each symptom with an appropriate remedy. Remember, however, the necessity for maintaining the strength of your patient, and of not interfering in any way with his nutrition. Remember, also, the importance of watching bronchitis in individuals with phthisical tendencies, especially when dullness on percussion and an alteration in the breathing denote the supervention of catarrhal pneumonia. If the plan of treatment I have indicated above be faithfully carried out, there is no reason why many patients with confirmed consumption should not enjoy a tolerable degree of health and comfort, even in our own climate, rigorous and changeable as it is.

In the management of the case before you, the occurrence of erysipelas, and the frequent repetitions of hemorrhage have, of course, necessitated the employment of remedies adapted to these conditions, but when they were not present the patient has taken chiefly tonics and nutrients.

There is one other point to which I wish to advert before closing this lecture, and that is in regard to the propriety of informing patients of their condition. It has been objected to this, that it would tend to produce depression, and that thus in many cases it might promote the extension of the disease. On the other hand, if patients are aware of the existence of the disease, or of a tendency to it, and if we take the trouble to explain to them its exact nature, they will frequently be able and more willing than they otherwise would be to carry out our directions intelligently; we should, at the same time, whenever it can be done truthfully, encourage them to look forward to a favorable result, which will counteract the bad influence of the information it has been our duty to give them.

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ACUTE PERITONITIS.

BY

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GENTLEMEN:—THE patient before you was admitted into the hospital on the 25th of the present month (two days ago). He is eighteen years of age, a cabinet-maker by trade.

At the time of his admission he stated that he had never been sick until three years ago last winter; then he had three successive attacks of intense pain in his abdomen, attended by nausea and vomiting of a greenish fluid, each attack lasting from two to three weeks. He does not remember whether the pain during these attacks was localized.

Each attack confined him to his bed for a week or two, and was preceded by obstinate constipation, which was relieved only by the free action of cathartics taken in large doses. Patient thinks he could always trace an attack to exposure to cold. Since the first attack he has suffered more or less with dyspeptic symptoms.

Last winter he had three similar attacks, and this is the third one he has had this winter. This season his first attack occurred in the early part of December, his second about Christmas, lasting about two weeks, after recovering from which he was in his usual health, able to perform his daily labor without inconvenience, had a good appetite, and suffered little, if any, pain in his abdomen.

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[*] Delivered in the amphitheatre of Bellevue Hospital, May 27th, 1875.

About three weeks before his admission to hospital, he worked all day in a cold shop with wet clothes, and became thoroughly chilled. For two or three days after this exposure he felt unwell, lost his appetite, had shooting pains in his abdomen, which were most severe about two inches below the umbilicus, radiating into the right and left iliac and hypochondriac regions. After remaining at home three days he returned to his work, still suffering more or less pain in his abdomen, with occasional nausea and vomiting.

Ten days before admission the pain in his abdomen became so severe, and the vomiting so constant, that he was compelled to keep his bed, then he first noticed that his abdomen was somewhat distended and exceedingly tender on pressure. The pain, vomiting, and tympanitis steadily increased up to the date of his admission.

Patient states that during this attack cathartics did not afford the relief experienced during previous attacks, in fact they seemed to make him worse. The day prior to his admission he had fæcal vomiting ; previously, the matter vomited had been fluid, of a yellowish-green color and bitter taste.

At the time of his admission, he was in a state of almost complete collapse ; face pallid, eyes sunken, conjunctiva injected, tongue red and dry, and lips parched. He complained constantly of thirst, yet rejected fluids as soon as they were taken into the stomach. His extremities were cold and moist, pulse thready in character and 120 per minute. Respiration thoracic, and 40 per minute. Temperature 101° F. Thighs flexed on the abdomen, abdomen distended and tympanitic, and, on pressure, exceedingly tender over its whole surface. Moderate pressure on abdomen very markedly increased the frequency of the respiration. Vomiting frequent and regurgitant in character. Micturition frequent, urine scanty, specific gravity 1021, alkaline, negative. Mind clear. Patient restless, but does not move the abdomen. Four ounces of brandy were immediately administered per rectum, and the opium plan of treatment was commenced,—ten minims of Mag. sol. morph. were administered hypodermically every hour until the patient was fully under the influence of the drug.

At 9 P. M., seven hours after the opium treatment was commenced, the respiration had fallen to 14 per minute, the pulse to 102, and the temperature to 100° F. The pupils were moderately contracted, the vomiting had ceased, the patient was free from pain, and although in a state of semi-narcotism he was easily aroused.

From that hour to the present time (42 hours) 5½ grains of morphine have been administered hypodermically. The respirations have never been above 16, nor below 12 per minute. The patient has been free from pain and has vomited but once. The pulse has ranged from 106 to 114 per minute. The temperature has varied from 99½° F. to 101° F.

In the treatment, our aim has been to keep the patient in a semi-narcotized state. Nourishment and stimulants have been administered in small quantities at short intervals.

As he now lies before you (in the amphitheatre), apparently in a deep sleep, from which he is aroused with difficulty, he is completely unconscious, for a fuller than usual dose of morphine was administered a short time before he was taken from the ward, in order that he might not be disturbed by the transfer. You notice that his face is flushed (the flushing is probably due to the opium), yet his countenance has a sunken, anxious expression,—the thighs are not flexed on the abdomen,—the respirations are 16 per minute, altogether thoracic,—each expiration is somewhat prolonged. The temperature is 100° F., and the pulse 118 per minute, small and firm.

As we examine the abdomen we find it greatly distended and tympanitic over its whole extent, except above the line of the ascending and transverse colon. The abdominal walls are firm and resistant, and as I make firm pressure with the palm of my hand over the central portion of the abdomen, although the patient does not open his eyes nor complain of pain, yet the respirations become accelerated to 22 per minute, and the pulse reaches 120. It is now ten days since the patient has had a movement from his bowels.

From the history which I have given you of this case, taken in connection with his present condition, although his symptoms are greatly masked by his condition of semi-narcotism (presenting, as he does, many of the unmistakable signs of opium poisoning), we are, I think, justified in making the diagnosis of *acute peritonitis.**

The first question that presents itself in the discussion of the many interesting points presented by this case, is in regard to its *etiology.*

In the present attack, the occurrence of fæcal vomiting proves, almost to a certainty, intestinal obstruction to have been the exciting cause of the disease. The cause of the intestinal obstruction is not so evident, but, from the history of his preceding attacks it would appear that the patient repeatedly had localized peritonitis, which must have given rise to more or less extensive peritoneal adhesions, and these adhesions are now causing partial or complete strangulation at some point along the line of the intestine.

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* This case terminated in recovery. Convalescence was fully established on the seventh day after his admission to the hospital. Up to that time 21 grains of morphine were administered hypodermically. The morphine was administered at intervals of from two to four hours, and during the seven days he was kept in a state of semi-narcotism. During the whole time, stimulants, milk, and eggs were administered in small quantities and at short intervals. On the sixth day the tympanitis began to subside, yet it was two weeks before it had entirely disappeared. He remained in the hospital about one month, and at the time of his discharge there was tenderness on firm pressure in the umbilical region, and a slight dullness on purcussion, which would indicate an abnormal amount of solid in the region of the mesentery glands.

Some of you may be asking the question,—May not the peritonitis in this case be due to the exposure to cold to which he was subjected at the commencement or just preceding the attack? In reply I would say: I do not believe exposure to wet and cold ever directly develops peritonitis.

Before proceeding farther in our clinical study it will be well for me briefly to enumerate the most commonly recognized exciting causes of Acute Peritonitis.

First to be named are intestinal obstructions and perforations. Under this head may be included typhlitis and perityphlitis, with ulceration; rupture of hepatic and other abscesses; ulceration and rupture of the stomach, of the gall or urinary bladder; hydatid cysts and aneurisms. In all these cases the inflammation spreads rapidly over the entire peritoneum.

Second: The extension to the peritoneum of inflammation from organs covered by peritoneum; this we find in those cases of general peritonitis, which are the result of the extension of inflammation from the uterus and its appendages, the liver, spleen, venous thrombi, typhlitis and perityphlitis, without ulceration. In all such cases the peritonitis remains circumscribed for some time after the commencement of the attack. When severe contusions, and penetrating wounds of the abdomen are causes of peritonitis, they must be studied from a surgical standpoint.

Lastly: In very many instances acute general peritonitis is the immediate result of an infection. Puerperal peritonitis, the peritonitis which develops in the course of an infectious disease, as pyæmia, septicæmia, etc., may be classed under this head. I shall not now enter upon the study of this latter form. It seems to me this should be regarded as a distinct type of the disease, having an entirely different history and treatment from that which is now engaging our attention.

There are many things of importance in the study of acute peritonitis, which this case would enable us to understand, which

I must pass over for the present. That I may impress you with the fact that the exact cause of peritonitis in any given case can with difficulty be determined during life, and with the hope that I may interest you in those things in the history and management of this disease, which have interested me the past few months, I will now relate the histories of several other cases which have come under my personal observation. I shall only relate such as terminated fatally, where autopsies were made.

On a certain evening, about 11 o'clock, I was called to see a well-nourished Scotch gentleman, sixty-one years of age, who up to that time had enjoyed such good health as never to have needed a physician. He was of very constipated habits, and it was his custom once or twice a week to take two or three dinner pills on going to bed. The afternoon of the day I saw him, he had taken his usual dinner down town at five o'clock, and immediately afterwards had walked from his place of business in Vesey Street to his home in Twenty-second Street. Just before he reached his home he was seized with a severe pain in the lower part of his bowels. As he entered the house he vomited, and by the time he reached the second floor he had an evacuation. The pain continued, increasing in severity until I saw him. At that time he was sitting in a chair, his pulse was 80 and normal, full and regular, and there was nothing to particularly attract my attention except an expression upon his countenance indicative of pain. On examination, I found that firm pressure upon the lower part of the abdomen, which was the seat of the pain, did not increase, nor did it relieve, his uneasiness. During the time I was making the examination he vomited the food which he had just taken. The abdominal muscles were not rigid ; the abdomen was soft over its whole surface. As I examined it carefully for peritonitis, I came to the conclusion that no peritonitis existed, and ordered the abdomen to be covered with warm fomentations, and ten drops of Mag. sol. morph. to be administered, to be repeated in two hours should the pain continue. At five o'clock in the morning, six hours from the time I first saw him, I was called to him again and found him in a state of collapse. The extremities were cold, pulse small and thready, and almost imperceptible, but intellection was perfect and there was no vomiting. The respiration was hurried, and, until a short time previous, the pain had increased in severity, although 60 minims of Mag. sol. morph. had been taken. In about one hour he died ; twelve hours from the commencement of the attack. The autopsy was made forty-eight hours after death. The body having been placed on ice was well preserved. On opening the thorax, the heart was found enlarged, its right side contained fluid blood. The aortic valves were calcified and slightly insufficient. The aorta was dilated and in a state of atheromatous degeneration. The lungs were normal, with the exception of the left apex, which was the seat of an induration. On laying open the abdominal cavity, the subperitoneal vessels were all intensely injected, and scattered over the surface of the peritoneum were flakes of coagulable lymph. The lymphy exudation was most abundant near the sigmoid flexure. The small intestines were carefully examined and found to be in a normal condition. On the sigmoid flexure, and on the descending colon, were diverticula, averaging three-fourths of an inch in diameter, standing out from the peritoneal surface. All of these were carefully examined, and were found filled with fæcal matter.

On dissection these diverticula seemed to be the result of hernia of the mucous membrane through the muscular coat of the intestine, the sac being formed of peritoneum and mucous membrane. The most careful dissection of these sacs failed to find any openings in either of them, but their peritoneal surfaces were the seat of the most intense inflammation, and from the more advanced stage of the inflammatory process at these points, they were undoubtedly the foci of the general peritonitis.

This case is interesting on account of the obscurity and rarity of its causation. I have carefully studied this subject, and find no recorded case where the peritonitis resulted from a similar cause. In the second place it shows the difficulty, and often in very many instances, the impossibility of determining, during life, the local cause of peritonitis, and this is often the case, even after death, unless great care is exercised in making the post-mortem examination. With a less careful post-mortem examination, this case would have passed for one of spontaneous peritonitis.

Again, the obscurity or the absence of the symptoms of peritonitis is a marked feature. Undoubtedly, peritonitis existed when this patient's pulse was eighty. When I first saw him, the only symptom that would lead to the suspicion of peritoneal inflammation was pain in the abdomen, and that was by no means characteristic; this fact, and others connected with its clinical history, will be more striking when I come to speak in detail of the symptoms of this disease.

A second case, equally obscure in its etiology, and interesting in its development, came under my observation in February of the present year.

A young man, 21 years of age (whom I visited in connection with Dr. R. O. Mason), was, at the time of the attack, in good health, of temperate and regular habits, and engaged in active business. He gave no history of severe illness since his infancy, yet always had had trouble with his bowels. On the 28th day of last February he ate a hearty dinner of indigestible food; among the articles taken he mentioned goose and pickles. Immediately after dinner he rode up-town (about three miles) on the front platform of a street-car. The day was cold and raw; he became somewhat chilled during his ride, and soon after his return to his room, about 10 o'clock in the evening, he was seized with vomiting, which continued at short intervals all night. The matter first vomited was undigested food, afterwards a greenish fluid.

He stated that his bowels were habitually constipated, and had not moved for forty-eight hours, although he had frequent desire to stool and had made several attempts since the vomiting commenced.

The patient lay indifferently upon his back or right side, with limbs extended, his face, although pale, was neither anxious nor expressive of severe pain. Pulse 98 per minute and full. He only occasionally complained of griping or colicky pain. This was frequently followed by vomiting, rather violent in character ; the matter vomited was simply such as had been taken into the stomach. There was no point of tenderness on firm pressure. At this time the diagnosis was gastric irritation from indigestible food, with constipation possibly from intestinal obstruction. Large enemas of water were ordered, and opium in sufficient quantity to procure sleep.

The following day the patient seemed very much better, had slept some, vomited at long intervals (four or five hours). Pulse 88 per minute. Bowels had not been moved, and large enemas were given with no effect, after which a hypodermic injection of one half of a grain of morphine was administered, which kept the patient free from pain during the night.

On the next day patient was not as well ; had a more anxious expression of countenance, and complained of a fixed pain in the umbilical region, which extended up to the right shoulder. There was tenderness and some dullness in the left hypogastric region, but the abdomen was not tympanitic. I then saw the case for the first time, and diagnosticated local peritonitis with intestinal obstruction, either at the sigmoid flexure of the colon, or in some of the small intestines in that region. The copious injections which had been given and retained, rather excluded the former supposition, and the latter was therefore accepted as the true one. The next day the peritonitis became general over the hypogastric region, although the opium plan of treatment had been pushed to semi-narcotism. Vomiting was frequent and stercoraceous in character, collapse occurred, and the case terminated fatally on the sixth day of the disease.

At the post-mortem examination, on opening the abdominal cavity, a considerable quantity of dark-colored serum was found, —the upper portion of the intestines was greatly distended,—the subperitoneal vessels were intensely injected, and numerous patches of fibrous exudation were found on the visceral and parietal surface of the peritoneum, especially on the left side. The large intestines were empty, with the exception of a few small fæcal masses in the cœcum and commencement of the colon. The ileum was nearly empty, was very small, and, at first view, presented the appearance of an immensely elongated vermiform appendix. The jejunum was unusually large, was distended with gas, and partly filled with fæcal matter in a fluid state. At its lower extremity, near its juncture with the ileum, a portion of the intestine, about four feet in length, was gathered into an irregular mass, and bound together, chiefly, but not wholly, along the mesenteric border, by firm fibrinous bands which were undoubtedly of long standing. The main points of adhesion of these fibrous attachments were included within a space of about four inches at the very extremity of the jejunum, and at the commencement of the ileum. The constriction of the intestine was complete, a firm fibrinous band girdling it at this point. Above this point, the jejunum was greatly distended and filled with fluid fæcal matter; while below the ileum was very much diminished in size, and empty. The mesenteric glands were markedly enlarged, and for some distance about the main point of adhesion, both in the free mesentery and in that which forms the covering to the intestine were the seat of calcareous degeneration.

In this case, without the post-mortem history, we should have regarded the peritonitis as unquestionably of spontaneous origin. A young man, in apparently good health, is exposed for a long time to a current of cold air across his abdomen, becomes chilled, and is immediately seized with vomiting and pain in his abdomen, followed by all the characteristic phenomena of peritonitis, and dies on the fifth day from the commencement of the attack. The calcareous changes in the mesenteric glands go to prove that the very extensive peritoneal changes of old date, which were found at the autopsy, and which were the cause of intestinal obstruction, must have existed from childhood; and yet, until the commencement of the fatal attack, there was no evidence that they had ever given him any inconvenience.

The slow development of the symptoms of general peritonitis is rather remarkable when we consider the completeness and extent of the intestinal obstruction. That acute local peritonitis existed at the time of the commencement of the attack there can be but little doubt, although it did not become general until forty-eight hours before death occurred.

Quite recently another case of peritonitis has come under my observation, which is equally instructive in connection with the etiology and symptomatology of this disease.

On the evening of the 27th of last month, I was requested to visit a merchant, 46 years of age, who, for the past eight or nine years, had been subject to mild attacks of bronchitis and follicular faucitis, and, on that account, had been under my immediate observation.

For the past four or five years he had been the intimate friend of a very intelligent physician, who had resided in the house with him, but, neither to him nor to myself, had he ever complained of any gastric or intestinal disturbance, nor did his family remember him to have ever manifested symptoms of such disturbance. He could not remember to have ever vomited.

He was free from any hereditary or acquired predisposition to disease, and, although apparently neither strong nor robust, was capable of undergoing great physical exertion without fatigue.

For five weeks preceding his last illness, he had been exposed, the greater part of each day, to the fumes of lead, but, apparently, it had had no injurious effect upon him, and at the time of his attack he was in his usual good health.

In the morning of the day on which I was requested to visit him he had felt a slight pain in the abdomen, resembling colic, not so severe, however, as to detain him at home.

I saw him about six o'clock in the evening; he had then a severe, lacerating pain, fixed at a point on a line with and about two inches to the left of the umbilicus, which, at first, was relieved by firm pressure. I found him sitting in a chair, with a countenance expressive of intense pain, and, on examination, found that there was a spot of tenderness over the seat of pain. The abdominal walls were flaccid. Pulse 70. Temperature normal. A slight blue line was noticed along the edge of the gums. I administered 35 minims of Mag. sol. morph. (within two or three hours) before he obtained relief from the pain. During the night he slept a disturbed sleep, at times complaining of pain. The next morning I again found him with a countenance expressive of intense pain,—pulse 80, and soft,—temperature normal,—abdominal muscles slightly rigid,—area of tenderness not increased. During my visit he vomited for the first time, the matter vomited was tinged with green. The diagnosis of peritonitis was made.

I placed him immediately on the opium plan of treatment, directing that morphine should be administered every two hours in sufficient quantity to keep the patient in a semi-narcotized condition. At seven o'clock in the evening his pulse had reached 120 per minute, and was firm and tense in character. Temperature, 100° F. Respiration, 20 per minute and thoracic. Abdomen considerably distended and tympanitic. Patient was easily roused and when moved complained of pain; had taken one quarter of a grain of morphine every two hours during the day. Ordered one quarter grain of morphine to be administered every hour during the night, unless symptoms of narcotism were developed. During the night there was little change in the patient's condition. Pulse remained at 120. Respiration never went below 16 per minute. General capillary circulation good; no vomiting; nourishment and a moderate amount of stimulants were freely taken. About ten o'clock the next morning the pulse became more frequent, reaching 140 per minute, and was irregular. Axillary temperature, 101° F. General capillary circulation imperfect. Extremities were becoming cold. Tympanitis increased. Vomiting persistent, and matter vomited "*spinach green*" in color. Commenced the administration of morphine hypodermically, and of stimulants by the rectum. At six o'clock in the morning he passed into a state of collapse, and died at four o'clock the following morning. Mind clear until a few moments previous to his death. Death occurred 56 hours after the commencement of the peritonitis. Autopsy was made six hours after death. On opening the abdominal cavity, the peritoneum lining the abdominal walls was found to be greatly thickened and covered by a thick exudative membrane. Both layers of the peritoneum were intensely congested, roughened, and covered in places with recent fibrinous exudation; the intestines were distended with gas. The stomach was found lower down and more to the left than usual, being bound in its abnormal position by adhesions, which were not readily broken down.

A perforation of the stomach, about the size of a three-cent piece, was found in the lower curvature near the cardiac orifice. On opening the stomach the mucous membrane was found to be greatly thickened and congested, and of a dark color. In the lesser curvature near the cardiac orifice was a large ulcer nearly the size of the palm of the hand, with thick inverted edges and surrounded by firm, hard nodules, which, under the microscope, proved to be round-celled sarcoma. The floor of the ulcer was made up of cicatricial tissue, except in the centre, where was the perforation. The pylorus and duodenum were dilated much beyond their normal calibre. The veins of the portal circulation were distended, other organs were normal.

In this case you can readily understand the impossibility of ascertaining during life the cause of the peritonitis; in fact, it could hardly have been suspected before the autopsy was made.

The fixed seat of the pain on a line with and two inches to the left of the umbilicus, would almost exclude perforation of the stomach, and yet the abnormal position of the stomach placed the perforation directly at the point where the patient first felt his pain. We are led to conclude that perforation did not occur until some time after the commencement of the attack, from the fact that the symptoms which marked the occurrence of the general peritonitis did not manifest themselves until some time after the occurrence of the intense pain which undoubtedly marked the localized peritonitis. On the other hand, the speedy fatal termination of the disease after the occurrence of the symptoms of general peritonitis would almost necessarily lead us to the conclusion that perforation at some portion of the alimentary track was the cause of the general peritonitis, for peritonitis resulting from perforation and the escape of intestinal gases into the peritoneal cavity, very markedly differs from general peritonitis resulting from the gradual extension of inflammation from an inflamed viscus or a local traumatic cause. The disease of the stomach, which led to the fatal accident in this case, must have been of long standing. We arrive at this conclusion not only from the amount of cicatricial tissue in and around the ulcer, but from the thickened and pigmented peritoneum in the region of the stomach, and from the firmness of the adhesions which bound the stomach in its abnormal position. These stomach changes may have occurred in childhood, and the perforation may have been merely the result of degeneration in the new cicatricial tissue from some not readily determined interference with its nutrition.

I will give you the history of one more case which came under my observation a few months ago, in order to show you the difficulties which sometimes attend the recognition of ulceration and perforation of the vermiform appendix as a cause of peritonitis. About ten o'clock one morning I was requested to visit a gentleman, thirty-four years of age, who had been ill during the previous night with diarrhœa. I found him seated at the breakfast-table, reading the morning paper. He complained of slight pain in the abdomen, accompanied by frequent watery discharges from the bowels. His pulse and temperature were normal, and as he stated to me that the evening previous he had eaten freely of "lobster salad," I regarded the case as one of simple diarrhœa, and prescribed accordingly. On the evening of the same day I was requested to visit him again. Then I found that his diarrhœal discharges had ceased, but that the pain in his abdomen had increased in severity, and was paroxysmal in character. On a careful examination of his abdomen, I found a limited and well-defined area of tenderness in the left iliac fossæ, and he stated that his abdominal pains seemed to radiate from this point. In reply to close questioning, he informed me that the only severe illness which he remembered occurred about ten years previous to this attack, while he was residing temporarily in Naples; then he was dangerously ill, and was confined to his bed six weeks; his physician called his disease "Roman fever." Since his recovery from that illness he had been perfectly well, and had prided himself on his ability to undergo prolonged and severe physical exercise without fatigue. Two evenings previous to his present attack, he had performed the most severe form of military drill for five consecutive hours without inconvenience or fatigue.

At my second visit his pulse was 76 per minute, and his temperature was normal. There was no tympanitis. He was cheerful; his countenance not anxious, and although I could not satisfactorily determine the cause of his abdominal pain, I excluded peritonitis for reasons already named. I ordered a full dose of opium and left him for the night, with little or no anxiety about his case. He slept quietly during the night, but on attempting to rise in the morning was again seized with pain in his left iliac region ; vomiting followed, and when I saw him about ten o'clock in the morning all the symptoms of acute general peritonitis were present. Although I brought him as speedily as possible fully under the influence of opium and kept him in a semi-narcotized state, in forty-eight hours he passed into a state of collapse, and died on the fourth day from the commencement of the illness.

At the post-mortem examination, made eight hours after death, the entire peritoneum, both visceral and parietal, was found intensely congested, and covered more or less abundantly with recent plastic material.

The inflammatory changes were most abundant, and in a more advanced stage, at the lower portion of the abdominal cavity. The intestines and pelvic viscera were firmly bound together by old adhesions. On attempting to remove them, an abscess was disclosed, which contained about a pint of thick pus. This abscess occupied the left side of the pelvic cavity, extending up into the left iliac fossæ. Evidences of recent peritonitis were most abundant over the iliac portion of this abscess. Anteriorly, the abscess was bounded by the bladder and a coil of small intestines ; posteriorly, by the rectum ; laterally, on the left side by the sigmoid flexure (which was displaced downwards), and by the wall of the pelvis, on the right, by the cœcum (which was displaced to the left of the median line and adherent to the left inguinal region). The walls of the abscess were thick and pigmented. The vermiform appendix was found elongated, and floating in the abscess, with its calibre enlarged, and a perforation near its cœcal extremity about the size of a five-cent piece, which was evidently the result of ulceration. Peyer's patches were elevated and pigmented. It is hardly necessary for me to state that in this case, I did not even suspect ulceration or perforation of the vermiform appendix to be the remote or immediate cause of the peritonitis; for the primary seat of pain and tenderness was in the left instead of the right iliac region, and there was no previous history of any trouble in the region of the vermiform appendix.

The suddenness of its development, its great extent, and its speedy, fatal termination, all indicated the occurrence of intestinal perforation and the escape of intestinal gases into the peritoneal cavity, but that the vermiform appendix occupied the left pelvic cavity, seemed almost improbable.

It is also somewhat remarkable that so extensive an abscess, evidently of long standing (as shown by its pigmented walls), could have existed in the abdominal cavity without giving some signs of its existence. The patient appeared to be in perfect health up to the time of the occurrence of the peritonitis.

It would be interesting to consider in detail the process by which the abnormal position of the intestines was brought about, but such a discussion does not come within the scope of our present studies.

You will have noticed that three of the four cases (the histories of which I have just given you) terminated fatally. It was on account of the post-mortem revelations, taken in connection with their clinical history, that I have detained you with their recital.

The statements and facts in regard to its etiology, which I have brought before you, must, I think, convince you of two things: First, that acute peritonitis is rarely, if ever, of spontaneous origin. Second, that frequently it is with the greatest difficulty we can determine its cause, although it is of the greatest importance, as on it depends our prognosis, and to some extent, the treatment of the case.

The morbid appearances presented at the autopsies of the three fatal cases already mentioned, give a very complete history of the pathological lesions of this disease. I will briefly review these in the order in which they appeared.

More or less redness of the peritoneum was present in all the cases mentioned, this was most marked in the first case. In each the redness was most intense at the point where the inflammation had its origin, and for the most part was due to hyperæmia of the capillaries of the serous and subserous tissue, somewhat to punctate spots of ecchymosis. In those parts where the inflammatory process was more advanced, the redness was least, and the peritoneum had lost its natural lustre, owing to the detachment of its epithelial covering, the peritoneal tissue was infiltrated, and presented a somewhat swollen appearance, and its free surface assumed a rough, shaggy appearance, which was due to an exudation upon its surface of a soft, red, elastic material ; this, in some instances, was spread over both the visceral and parietal surfaces of the membrane, and in others, it occurred in small patches.

In each case, as the abdomen was opened, a layer of yellow material was found covering and agglutinating the intestines more or less extensively. This material was coagulable lymph, which had been poured out from the serous and subserous vessels, and had collected upon the free surface of the peritoneum. It enclosed in its meshes an innumerable number of young cells, which were either leucocytes or changed epithelial cells.

In the two last cases, serous effusion was found in the most depending portion of the abdominal cavity. It was not clear, but contained flocculi of lymph cells and free nuclei.

In the second case, the cell formations were abundant, giving the effusion all the characteristics of pus.

In very recent peritoneal inflammation (as was very plainly shown in the fourth case), when you remove the plastic exudation from the free surface of the peritoneum, you find immediately underneath it a layer of embryonic cells in a condition to develop into new connective tissue. It would appear that as soon as the peritoneum is denuded of its epithelium, a layer of new cells are developed, which may enter into new connective tissue formation. The nature of the subsequent changes will depend upon the intensity and duration of the inflammatory process. If adhesions take place between contiguous layers of the peritoneum they are effected in the following manner: the new cells imbedded in the fibrinous exudation become elongated and spindle-shaped, and form connective tissue cells, the fibrine fibrillates, and a net-work of capillaries permeates the young false membrane. In some instances the adhesions take place by the formation and growing together of papillary outgrowths from the sub-epithelial tissue.

The serous, fibrinous, and cellular exudations may all undergo absorption, or the serous exudation may be absorbed and the plastic exudation may agglutinate the two peritoneal surfaces. Usually, if recovery takes place, all the exudation upon the peritoneal surfaces, and that exudation which gravitates to the most depending portion of the peritoneal cavity undergo absorption.

Whenever two surfaces of the peritoneum become bound together by firm adhesions—the adhesions take place either between the abdominal walls and the intestines, or between folds of the intestines—you may have the bands of adhesion forming bridles, which may constrict a loop of intestine, and thus give rise to the intestinal obstruction which may be the exciting cause of a fresh peritonitis. This is probably the condition in the patient before you. The second case which I related was a marked example of this, as shown by the autopsy.

In the patient before you, and in all the cases I have related to you, the disease commenced with a most intense pain, which, for a short time was fixed at one point in the abdomen; for a time the seat of pain could be covered by the ends of the fingers. In each case it spread more or less rapidly, so that in from six to twenty-four hours it occupied the whole region of the abdomen.

In most of the cases to which I have called your attention, the constitutional symptoms were developed slowly, and the pulse was not increased in frequency until some time after the commencement of the attack. The tenderness on pressure was first local and then general; throughout the disease the tongue was covered with a white coating, and the countenance early assumed an expression of anxiety.

In the first and third cases, where the peritonitis resulted from perforation and the escape of intestinal gases into the peritoneal cavity, the first symptom was excessive pain over the whole abdomen, attended by great general prostration.

When peritonitis results from infection it is always ushered in by a severe chill, followed immediately by intense fever; but, in the form of peritonitis which we have been studying a chill is rarely present.

In whatever manner peritonitis may be ushered in, it is evident that pain is one of the earliest and most constant symptoms. Ordinarily, by the time you reach a patient with acute peritonitis you will find him suffering severe cutting pain in the abdomen, somewhat paroxysmal in character, and the pain will be greatly increased by firm pressure. In a few rare instances, acute peritonitis is unattended by pain. A patient with peritonitis does not toss about in bed, but lies on his back with his knees drawn up so as to relax the abdominal muscles. His abdominal respiration is arrested and his breathing is altogether thoracic. Whenever it is necessary for you to explore the abdomen by pressure, take care not to make your examination unnecessarily painful; press gently with the open flat hand, watching closely the countenance of your patient, and if you are causing pain, either the expression of the countenance, or, as in the case before you, the increase in the frequency of the respiration will indicate it, even though the patient may not have complained.

As you notice, the abdomen of this patient is distended, tense and, on percussion, we find it tympanitic. This tympanitis is caused by the filling of the intestines with gas; the gas is never in the cavity of the peritoneum. As the disease advances the tympanitis increases, the abdomen often becoming greatly distended. Whenever a peritonitis has extended over the whole peritoneal surface, excessive tympanitis is developed; so long as the peritonitis is localized the tympanitis is usually slight. As the disease advances the tympanitis increases until, in some cases, the intestines become greatly distended and interfere with respiration, increasing the cyanotic condition, and, by the tension of the peritoneum which they cause, increase the peritonitis. Excessive tympanitis must always be regarded as an element of danger.

Vomiting is another prominent symptom of peritonitis. It was present to a greater or less degree in all of our cases. Usually, it does not come on until the second day of the disease, but in the first and second cases mentioned it was present at the very outset of the disease, preceding the pain. At first, the matter vomited is merely the contents of the stomach, but at some time in the course of the peritonitis it will assume a green color, like "boiled spinach," this is characteristic of the disease, at least if I should notice this peculiar looking matter vomited by one who was not even suspected of having peritonitis I should be almost certain of the existence of the disease.

Whenever stercoraceous vomiting occurs in the course of a peritonitis, either as an early or late symptom, you must recognize the existence of intestinal obstruction; such obstruction as is shown in the second case brought to your notice, is usually the cause of the peritonitis. In a few cases of intestinal obstruction, the vomiting of fæcal matter will precede the development of the peritonitis.

As patients with peritonitis reach the advanced stages of the disease, and the serous covering of the stomach is involved, the stomach will immediately reject everything that is swallowed. The boy before you, when he was admitted into the hospital, was vomiting constantly, so much so that it was necessary to administer morphine hypodermically, and brandy per rectum. As these patients approach their end, the serous covering of the diaphragm sometimes becomes involved, hiccough is then apt to accompany the vomiting, which becomes regurgitant in character, and the matter vomited has the appearance of coffee-grounds, showing that there is capillary hemorrhage from the mucous membrane of the stomach.

In all our cases constipation was present throughout the whole course of the disease. This is the result of complete arrest of the peristaltic action of the intestines caused by the extension of the inflammatory process from the peritoneum to the muscular coat of the intestines. So long as inflammation of the intestinal peritoneum continues there can be no movement of the bowels, notwithstanding you administer irritating cathartics.

As I have already stated, if the peritoneum covering the diaphragm becomes inflamed, hiccough is present, and sometimes becomes very distressing ; it was present in the case before you, before the patient was fully brought under the influence of opium. Vomiting, although always present, does not become a distressing symptom unless the serous coat of the stomach is involved. If the peritoneum over the bladder becomes either primarily or secondarily involved, there will be more or less strangury.

I stated that as the disease advances, and the intestines became distended with gas, percussion elicits a tympanitic note over the whole of the abdominal cavity. In some cases, however, there will be a rapid effusion of serum, which will gravitate to the most depending portion of the peritoneal cavity, and then there will be a line of dullness corresponding to the position of the fluid, which will change with a change in the position of the patient. If a large amount of coagulated lymph has been poured over that portion of the peritoneum which covers the liver or spleen, a distinct fremitus will sometimes be communicated to the hand as it passes over that portion of the abdomen, and auscultation may discover a creaking, friction-sound with each respiratory movement, which resembles the friction-sound of pleurisy. This friction-sound for a time was present in the third case mentioned. In each of the fatal cases, as the peritonitis advanced to its termination, the pulse became quick and thready, the countenance pinched and ghastly, the surface cold and clammy, and was covered by a more or less profuse perspiration, the breathing was accelerated and labored, the capillary circulation of the surface was interfered with, and the lips and fingers became blue (in the fourth case, the hands and feet were cold and blue twelve hours before death occurred) ; finally the patient passed into a state of complete collapse, the mind remaining clear and collected until death occurred. After peritonitis becomes general, its progress is uniform, and unless arrested by treatment, it marches steadily on to a fatal termination. Its duration is from three to eight days. In all cases, to a great degree, the prognosis is determined by its causation. When the peritonitis is excited by the escape of intestinal gases into the peritoneal cavity, the prognosis is uniformly bad. This class of cases usually very rapidly terminates fatally. When the peritoneal inflammation results from the extension of inflammation from some abdominal viscus, if the case is seen early, before the peritonitis becomes general, the prognosis is good. In the majority of cases, even general peritonitis, occurring under such circumstances, can be controlled.

As regards its differential diagnosis, in an advanced stage this disease will rarely be mistaken for any other affection ; but at its commencement, while it is limited to a circumscribed space, its symptoms may be so masked and confounded with the symptoms of other morbid conditions as to render the diagnosis very difficult, and even a careful diagnostician will often make serious mistakes.

The diseases which it most resembles, and which are most likely to be confounded with it are : First, inflammation of the mucous membrane of the small intestine, or enteritis ; second, colic ; third, the passage of a biliary or renal calculus ; fourth, rheumatism, neuralgia, and hysteria ; fifth, lead poisoning.

I will briefly consider the points of distinction between these affections and peritonitis.

The following are the principal points of difference between inflammation of the mucous membrane of the small intestines and peritonitis. Enteritis always commences with diarrhœa ; while in peritonitis, as a rule, the bowels are constipated. (In the fourth case mentioned, there was diarrhœa present for a few hours after the commencement of the attack.) Again, vomiting, although it may be present in simple intestinal inflammation, is not so severe as in peritonitis, and the matters vomited have not the characteristic spinach-green appearance of peritonitis. There is little or no tympanitis in enteritis, while increasing tympanitis is a prominent symptom of peritonitis. Pain is not so intense in enteritis as in peritonitis, and is temporarily relieved by firm pressure, while firm pressure increases the pain in peritonitis. The pulse is not so firm, hard, nor tense in enteritis as in peritonitis.

Peritonitis and colic have the following points of distinction: In colic, the pulse is not altered, the pain is relieved by firm pressure, the abdomen is seldom much swollen, there is little or no constitutional disturbance. Sometimes colic comes on as the herald of a more grave disease, and ends in the development of peritoneal inflammation. In *colica pictonum*, the ordinary symptoms of colic are present ; there is constipation and abdominal pain, which is frequently very violent in character. But with these there are other symptoms, such as pain in the head and limbs, a blue line along the edge of the gums, and loss of power in the hands and forearms. These symptoms, and the fact that there has been exposure to the poisonous influences of lead, will lead one to a correct diagnosis. Rheumatic affections of the abdominal muscles sometimes render the abdominal muscles excessively painful, so that moderate pressure causes great suffering, and, notwithstanding cases of acute peritonitis occasionally occur which have a rheumatic origin, yet they are so rare, that ordinary care will prevent any mistake in diagnosis. Under such circumstances, negative facts will be our chief guide. In such cases the pulse is little affected, nausea and vomiting are not present. The countenance has not the anxious, pinched expression of peritonitis, and if the abdomen is carefully examined the pain will be found most marked at the origin and insertion of the muscles. Besides, on careful questioning, it will be found that other parts of the body are subject to rheumatic developments.

The pain of abdominal neuralgia is described as a tight girdle passing around the body,—it traverses the course of the genito-crural nerve. There is tenderness along the spinal processes, and the legs and genito-urinary organs are more or less affected. Again, there is no tympanitis nor pain on pressure, the pulse is not accelerated, there are no evidences of collapse, and there is absence of the other phenomena so expressive of peritonitis.

In hysteria, the patient is ready to complain of increased pain almost before the hand has touched the abdomen, and when pressure is really made, it does not increase the pain, as it does in peritonitis. The pulse is natural, the tongue clean, the countenance is not expressive of acute disease, the breathing is not thoracic, and on inquiry, you will find that large quantities of colorless urine have been voided.

In the majority of cases the passage of calculi can be recognized without difficulty. The passage of biliary calculi is always attended by pain in the epigastrium which shoots directly back to the spinal column. Pressure is not painful unless it is continued for a long time. There is little change in the pulse. For a time the patient's agony is intense, then he is suddenly relieved of all pain. If vomiting occurs, he vomits only the contents of the stomach. Usually, within twenty-four hours from the commencement of the attack, a yellow tinge of the surface may be observed.

In the passage of a urinary calculus, the pain passes from the back round to the abdomen, and along the course of the ureter on the affected side, producing (in the male) retraction of the testicle on that side. The pulse is not accelerated unless the attack is prolonged. Bloody urine is a common accompaniment.

Acute peritonitis is a severe, rapidly progressing, and dangerous inflammation, and, on this account, it has always been treated heroically by the profession. Until quite recently, all patients with peritonitis were subjected to excessive and repeated bleedings. Tart. emetic was administered in nauseating doses to prolong the effects of the bleedings, and as an adjunct to these, calomel was administered for its specific constitutional effect. At the same time many physicians of recognized authority were very urgent to obtain the purgative effect of cathartics, and for this purpose recommended and administered large doses of powerful drastic purgatives. They maintained that if you could only move the bowels of a patient suffering with peritonitis, he would certainly recover. In a certain sense this statement is true, for so long as peritonitis exists to any extent, it is impossible to move the bowels even by the administration of the most powerful purgatives. The peritoneum and the muscular coat of the intestines are nourished by the same capillary vessels, therefore, if the peritoneum be inflamed, the muscular coat of the intestines will also be inflamed, and, as a result of the inflammation, the muscular fibres of the intestine will become paralyzed, and their peristaltic movement arrested ; and this arrested peristaltic movement cannot be excited by any amount of irritation applied to the mucous surface of the intestines. Do not, therefore, under any circumstances administer purgatives to patients with acute peritonitis.

The plan of treatment which I have adopted in the management of the case before you is very unlike any of those plans to which I have referred ; a plan which gains in favor with me with every year's experience, and which, if commenced early, in the majority of cases, is successful, unless the peritonitis is due to perforation and the escape of gases into the peritoneal cavity, or to complete and permanent intestinal obstruction. At least, I have found it rarely to fail in accomplishing desired results when commenced within twenty-four hours after the advent of the disease, and pursued without interruption. This plan has been termed the "opium plan of treatment." We are indebted to Prof. Alonzo Clark, of this city, for developing it and bringing it prominently before the profession; although, strictly speaking, the use of opium in the treatment of peritonitis cannot be said to have originated with Prof. Clark. The details of this plan are as follows:

As soon as you have decided that your patient has acute peritonitis, administer at once from two to five grains of solid opium, or from one-half to a grain of morphine. The exact quantity in each case is to be determined by the condition of your patient. As a rule, to a strong, vigorous male, you may give four grains for the first dose; to a feeble female it is not well to commence with more than two grains.

In the treatment of this disease, you will observe how greatly pain and inflammation modify the effects of this powerful drug. I have administered to patients with peritonitis four grains of opium every two hours for twenty-four hours, and then have obtained only a moderate effect of the drug. The point which you wish to reach in the administration of opium in this disease is moderate narcotism, in which state you must strive to keep your patient, not only until all pain and tenderness has subsided, but until the pulse has reached a normal standard and the tympanitis has entirely subsided.

This brings us to the question,—What are the indications which are to govern you in the administration of each dose of opium? You must be prepared, at the commencement of your treatment of a case of peritonitis according to this plan, to be present and decide upon the quantity of opium to be given at each dose, until your patient has fully convalesced. You cannot trust attendants, however intelligent they may be.

As you bring your patient fully under the influence of the opiate, you will notice that the entire surface of the body becomes bathed in a profuse perspiration; in twenty-four hours a rash, due to the opium, will make its appearance on the face and neck; this is accompanied by an itching of the surface and a constant disposition to rub the nose. The pupils become contracted, the eyes suffused, the countenance assumes a dull expression, and there is a constant irresistible disposition to sleep. The pulse becomes lessened in frequency and force, and the respirations, which before the administration of the opium may have ranged from 40 to 60 in a minute, as the patient comes fully under the influence of the opium, become less and less frequent, until they are only 12 in a minute. Now, exercise great care in the administration of your opium,—you have your patient in the condition in which you wish to keep him. By holding him in this state of semi-narcotism, you will accomplish all that can be accomplished by the opium plan of treatment, and with the respiration at twelve in a minute, you are perfectly safe. The amount of sleep is not to be taken into account, but the profoundness of the slumber is of great importance. If you find it difficult to arouse your patient, you must stop the administration of the opium. If by mistake or negligence your patient becomes fully narcotized, the respirations will sometimes diminish in frequency to seven or even five in a minute. In this extremity, if the administration of the opium be stopped, usually after a few hours your patient will rally from its effects, but avoid extremes, endeavor to keep your patient in a quiet sleep, not profound, but one from which he may be easily aroused. When the pulse begins to diminish in frequency, and becomes fuller, you may be certain that you are controlling the peritonitis, and as it is controlled your patient will become more and more susceptible to the influence of the opium. Slowness of respiration and absence of pain cannot be relied on as sure indications that the opium is controlling the inflammatory action, but a diminution in the frequency of the pulse and a subsidence of the tympanitis are sure indications that the peritonitis is arrested, and that ultimate recovery is probable.

In most cases, if an acute peritonitis does not depend for its exciting cause upon the escape of intestinal gases into the peritoneal cavity, or upon complete intestinal obstruction, you can control the inflammatory action within forty-eight hours from the commencement of the attack, by adopting within twelve hours this plan of treatment. You must, however, continue the treatment four or five days longer, for there is still danger from a renewal of the inflammation. As the condition of your patient demands less opium, you may diminish the dose, or increase the interval between the doses. A safe rule by which to be guided, is that so long as any tympanitis exists the opium plan of treatment should be continued. When convalescence is fully established, be not too anxious to overcome the constipation which usually exists, for a free spontaneous movement of the bowels generally follows a complete subsidence of the peritonitis. Wait at least a week for this result before you administer a cathartic, and then, if necessary, employ one mild in its action, such as castor oil.

In the case before you, warm poultices over the abdomen are the only local applications which have been employed. It is claimed by some that cold compresses have a much more beneficial effect than warm applications when applied over the abdomen. My experience leads me to doubt the utility of the former, while the latter are far safer, and, I believe, equally efficacious.

I have stated to you that when the peritonitis becomes general, excessive gaseous distention of the intestines occurs, and this distention greatly increases the danger to the patient; under such circumstances, I have recently resorted to minute puncturing of the distended intestine with a hypodermic or a very small aspirating needle, and thus relieved the intestinal distention by allowing the gas to escape. By so doing not only is the tension of the peritoneum (which becomes an exciting cause of the peritonitis) relieved, but the principal obstruction to the respiration is removed, and thus the cyanosis becomes less marked.

Immediate and marked relief is afforded by such a procedure, and as, thus far, I have seen no bad results follow, I am disposed to resort to it in all cases where the abdomen becomes excessively distended and tympanitic.

I remember one case in which the gaseous distention was excessive, and the peritonitis was supposed to be due to strangulation of a portion of intestine from old peritoneal adhesions, and the relief of the distended intestine by puncture was soon followed by a removal of the intestinal obstruction, and the rapid recovery of the patient. From this circumstance I can readily understand how a portion of intestine that was partially constricted by a band of adhesion might become completely obstructed at the point of stricture by a rapid gaseous distention of the intestine above the point of constriction, and the relief of the intestinal distention by puncture would very likely liberate the constricted portion, and thus overcome the strangulation, and so, perhaps, save the life of your patient.

The necessity of absolute quiet, and the frequent administration of nourishment and sometimes of stimulants, in small quantities, to this class of patients, is apparent. It is unnecessary longer to detain you with details in regard to treatment.

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ON GLEET; AND ITS RELATIONS TO URETHRAL STRICTURE.

BY

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THE secretion of the urethral mucous membrane serves as a protector, and lubricant, for the preservation of this membrane from contact with the irritating urinary fluid. It is made up of germinal granules—particles of bioplasm (Beale), which rise up through the interstices of the sub-mucous cellular tissue,* are transuded through the basement mucous membrane, and become organized as the protective and lubricative epethelial cells of the urethral mucous membrane; and, where the conditions of its evolution are in every respect perfect, in quantity just sufficient for the lubrication and protection of this structure. This is never sufficient to be perceptible to the naked eye, except as a moist glazing of the surface. Any excess is always the result of an abnormal stimulation of the natural processes, except in a single instance, purely physiological, when it proceeds from an erotic excitement, and appears at the urethral orifice as a transparent mucous exudation, which passes off with a cessation of the nervous impression which provoked it. The causes which unduly increase the secretion of this membrane (and in speaking of the urethral mucous membrane

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* Rindfleisch, Pathological Histology, Am. Ed., pp. 43, 99, *et seq.*

I include the glands, crypts, and follicles, made up of its local reduplications), are to be divided into two classes, viz.:—first, active inflammation set up by contagion, or clap ; and second, mechanical injury or obstruction, such as urethritis, from lodgment of calculus, or injuries caused by irritant injections, or instrumental violence, or from urethral stricture.

The first effect of an approaching inflammation of mucous membrane is an increase in the natural secretion. The mucous cells are hurried along, through their different stages of development, and, as the amount of secretion increases, it is less and less perfectly elaborated ; the germinal material is drawn to the surface with increasing rapidity, until cells, which, in health, pass through a gradual development, from the germinal granule to the fully-formed epithelial scale, now appear as a mass of emasculated corpuscles ; *pus cells*, which constitute what we are accustomed to designate as a purulent discharge.

The inflammation is thus characterized, during its continuance, whether arising from contagion or from mechanical or traumatic causes. The character of inflammation in the urethral mucous membrane varies in *degree*, rather than in kind. Its products are, to all appearance, similar, whether the result of gonorrhœal contagion, or from injury caused through instrumental or mechanical interference alone. The duration of the inflammation varies, as the cause is more or less vicious in its onset, more or less persistent in its influence. An inflammation set up by a gonorrhœal contact will continue, in spite of the most efficient and judicious treatment, for several weeks, while the inflammation caused by the forcible introduction of a sound, through a narrow meatus urinarius, *may* subside in a few days, and yet circumstances, *wholly unconnected with contagion*, may elevate this latter discharge, from a purely traumatic inflammatory product, so that it may communicate a disease to a perfectly healthy individual, in no way distinguishable from a gonorrhœal inflammation.

An inflammation, set up by contact with pus, from an acknowledged gonorrhœa, *at once* partakes of the vicious, contagious character of the inflammatory products from which it was derived. A simple urethritis *may* continue simple, and recovery take place within a short period, or it *may* be aggravated by various influences, such as vinous or sexual excess, contact with uterine or vaginal secretions, prolonged physical exercise, or from simple mechanical irritation, in a strumous or gouty diathesis, until it shall have *acquired* the property of contagiousness. Arrived at that point, urethritis of non-venereal origin, does not differ in any way from that which has been originally acquired by contagion. The *contagium*, or contagious element present in gonorrhœal inflammation, would seem to be due to an acquired viciousness, from the fact, that this *contagium* may be developed, or induced, in simple urethritis, by the various causes above enumerated, independently of contact with the gonorrhœal secretion. This position, *most important in practice, as well as in a medico-legal point of view*, is capable of substantiation by eminent authority, and besides, I have personal knowledge of its truth, from a number of carefully observed and recorded cases. The active stage of an inflammation of the urethral mucous membrane is called an *urethritis*, when resulting from causes independent of venereal contact, and when acknowledging a contagious origin, it is termed a *gonorrhœa*. Its duration in the great majority of cases, may be set down as four or five weeks. In the cases where complete recovery does not take place within this time, there is usually a subsidence of the more acute symptoms, and the case is then characterized by a painless or nearly painless discharge, more or less profuse, more or less purulent, which persists, in spite of the most earnest and judicious treatment by internal and local remedies, for weeks, perhaps months—often years; at times reduced to a mere secretion, which sticks the lips of the meatus together, when, upon a slight indiscretion in diet, a little sexual or vinous indulgence, and within a few hours it may return as a free, and possibly painful purulent discharge. This chronic form of urethritis, which has from time immemorial afflicted humanity, and which has probably been the source of more trouble, to patients and surgeons, than any other known difficulty, is familiarly known as GLEET.

It is usually either considered as a sort of chronic gonorrhœa, and treated on the same general principles, by internal remedies, and local injections, or is looked upon as the result of a debilitation of the urethral mucous membrane, which has no specific or contagious property associated with it, and is treated by specific and local means, with the addition of some constitutional remedies, addressed to the condition or diathesis, upon which the continuance of the difficulty is supposed to depend. Now, if it can be established that gleet is the result of a *mechanical* condition—that it may be produced, without the previous occurrence of a gonorrhœa, by a simple obstruction to the free discharge of urine through the urethra, and that this obstruction may occur as a result of *any* inflammation or injury, which shall implicate the submucous urethral tissues, it will then be clear that no treatment, which is not based upon the detection and removal of the mechanical difficulty, can be more than palliative. And if it can be shown that the detection of *contraction* is possible *in all cases of gleet*, and that its removal *is certain* to result in the cure of the gleet, the proof of the non-specific character of gleet may be considered established.

Mr. Henry Dick of London (whose brochure on the "Pathology and Treatment of Gleet,"* is in my opinion the most valuable contribution to the literature of this subject in any language), says "Gleet is always the consequence of a clap. I have never seen it idiopathically appear without clap, except in cases of disease of the prostate gland or the bladder. I would not say that idiopathic gleet never exists, but I have never seen it." This statement conveys the impression which is generally accepted by the profession in regard to the cause of gleet.

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* Published by Baillière Bros., in 1858.

Acute urethritis, from whatever cause, may be stated as a self-limited disease ;—a disease which, under various methods of treatment by internal remedies, such as copaiba, cubebs, sandal oil, etc., by alkalies and diuretics of various kinds, by local injections, such as sulphate of copper, sulphate of zinc, acetate of copper, acetate of zinc, acetate of lead, nitrate of silver, any and all of the mineral salts or vegetable astringents, preparations of carbolic acid, liquid glass (silicate of soda), fuller's earth, or any one of the thousand injections which have been used and lauded for their curative influences on acute urethritis,—or by no treatment at all,—has a tendency to get well within a limited time, and that time may be stated to be about four weeks. Dr. Bumstead* formulates the experience of the profession, in the past and present, in the statement that the average duration of the disease is three or four weeks. "*Greater success on the average,*" says Dr. Bumstead, "*is probably not attainable by any means with which we are at present acquainted.*" I have met quite a number of well authenticated cases, where there was a history of a severe gonorrhœa with inflammatory complications, which recovered within this time, under the use of *baths alone* —others, where homœopathic treatment was resorted to—and others again, where *no treatment at all* was had, and where recovery came within the four weeks. Now, while I am sure that a variety of remedies, local and general, may, when judiciously employed, enable the patient to pass through the disease with much more comfort, and less danger of subsequent trouble, than without treatment, yet I am quite prepared to state it as my opinion, based upon a large personal experience in the treatment of this disease, by the most approved methods, *that it is a self-limited disease in its acute form,* and that when it lasts longer than four weeks, or when apparent recovery takes place, and the discharge breaks out afresh, without new exposure, that *there is a complication present* (either the result of the current inflammatory trouble, or from some inflammation antecedent to the attack), which causes the continuance of the trouble, and which must be appreciated and removed before any *permanent* cure can be had. This complication is URETHRAL STRICTURE. Stricture in the sense of an abnormal contraction of the urethral calibre, at some point between the meatus urinarus and the bulbo-membranous junction, and I will furthermore state it as my conviction, that the continuance of the inflammatory trouble (and whenever there is an urethral discharge there is incontestably more or less inflammatory trouble) is due to the irritation kept up by the arrest, more or less complete, of the stream of urine, at the point of stricture, during the act of urination ; and by the imperfect emptying of the urethra after urination. *Chronic gonorrhœa—Gleet,* also variously designated as prostatic, gouty, scrofulous, is dependent, as a rule, on abnormal contractions of the urethral canal. The only exception that I recognize (aside from the presence of polypoid, or warty growths in the urethra) is the engagement of urethral sinuses, as the lacuna magna, or some one of those occasionally met near the meatus, possibly deeper down, and these I have never found engaged, unless more or less co-arctation at an anterior point was also present. *Chronic urethral discharge means stricture.* I am quite well aware, that well-defined stricture may be present, without a palpable discharge ; there is always to be found evidence of a certain degree of irritation present in all such cases, but there may be no appreciable discharge. When, however, there is *discharge,* there will, in every case, be found, if the examination is efficiently made, a *well-defined* and *unmistakable* point of *stricture.*

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* Bumstead on Venereal Diseases, Phil., 1870, p. 92.

The dependence of continued inflammation in gonorrhœa, and of the continuance of chronic urethral discharge, upon the presence of stricture, is no new discovery. All the recent approved authorities recognize it. Dick was the first, as far as I know, to insist upon a thorough examination of the urethra, for obstruction in every case of gleet, and his instructions for the examination of the urethra with the bulbous bougie of Le Roy D'Etiolles are minute and complete. Sir Henry Thompson says in his work on stricture of the urethra, page 90: "I have known instances in which this symptom (gleet) has been so prominent that the patient has been treated for a gonorrhœa, during a period of many weeks, without suspicion arising, that a stricture existed, which was its sole cause; the subsequent recognition of the contraction and its cure, having been attended with the complete cessation of the discharge.

Dr. Bumstead (Bumstead on Ven. Dis., 1870, p. 93), says: "It is not impossible that there is stricture of the urethra, which is the most frequent cause of the continuance of a gleety discharge following an attack of gonorrhœa."

Van Buren and Keyes, p. 71, say: "The most common of all causes for continued gleet is stricture, already present or forming," and yet in spite of the unmistakably pointed and positive statements of these, and other valued authorities, the usual treatment of chronic gonorrhœa and of gleet at the present day, is by *nostrums*. Sandal oil, copaiba, urethral injections in multiplicity, and the use of medicated bougies and sounds. And why? It is not that urethral stricture is doubted as a possible factor in the case; it is not that this is unrecognized as the most *probable* cause of the difficulty; but because the examination of the diseased urethra is conducted with *imperfect instruments*, and that as a consequence, no exhaustive examination of the canal is made. *The least contraction at any point in the urethral canal has been demonstrated as capable of causing the indefinite continuance of an urethral discharge, and even of establishing it,* de novo, *without venereal contact.*"* If this is the fact, then some means for the detection of the *least* contraction of the urethral canal must be used in order to ascertain the presence or absence of stricture. To this end, the first step must be to ascertain the *normal* urethral calibre in the presenting case. It has been proven that every urethra is an *individuality*, and that no *average standard* is of use in examining a given urethra. The establishment of the normal calibre is the first step towards ascertaining whether or no there be any co-arctations in its course. This can only be accomplished by *actual measurement*, and by an *urethra-meter*. The proposition is a purely mechanical one. Given a tube, urethral or otherwise, in which it is desirable to ascertain whether or not there exists a contraction of its *calibre* at any point, the first question to settle is *the size of the tube*, this effected, the determination of any *variations* becomes easy—without it impossible. The bulbous bougie was relied upon by Le Roy, Dick, and others, many years since, and it has been growing in favor very slowly but surely, so that now it is an indispensable instrument in urethral examination for stricture. Explorations with an ordinary sound, catheter, or straight bougie, are practically valueless in determining the size, locality, and number of strictures in a given case. The presence of a contracted meatus (one of the very common complications, as a result of infantile balanitis or gonorrhœal inflammation) makes the detection of any deeper stricture, if of greater calibre, quite impossible. With the bulbous sound or bougie, of a size, which, by firm but gentle pressure, may be made to pass through the meatus, its sudden release as it slips into the fossa navicularis, indicates that contraction is present at that point, but the release of the contraction becomes a necessity before the deeper canal can be efficiently explored, or the normal calibre of the urethra be estimated. It is here that

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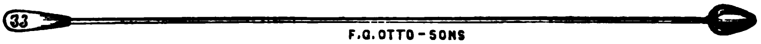
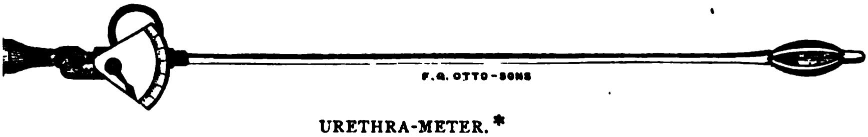
* F. N. Otis on Chronic Urethral Discharges, N. Y. Med. Journal, 1870, June.

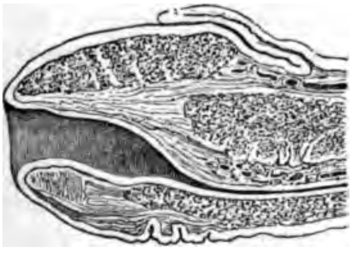


BULBOUS SOUND.

the value of the urethra-meter in the diagnosis of strictures becomes evident. This should be introduced through the contracted meatus (when this is not below 12 F.), and down to the bulbo-membranous junction. At this point the bulbous portion of the instrument is to be expanded, by means of the screw at the handle, until a feeling of fullness is experienced, when, if there is no stricture at the point of trial, the hand on the dial plate will indicate, with sufficient certainty, the normal calibre of the urethra under examination. Now, drawing the instrument slowly out, if stricture is present, the bulb will be arrested at that exact point. The screw is then turned, diminishing the size of the bulb, until it slips through the co-arctation, when a glance at the dial will show the calibre of the stricture. This subtracted from the figures indicating the normal calibre, will give the *precise value* of the contraction. The remainder of the canal examined in the same way, brings the bulb finally to the meatus, where, in the same manner, the greater or less degree of deviation from the normal size will be shown. Henle[†] has demonstrated the meatus urinarius to be of uniform size with the fossa navicularis, and thus from an anatomical stand-point, has demolished the error which has been



URETHRA-METER.*
(Improved.)



Vertical Section of the Anterior Portion of the Penis.—*Henle*.

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[*] The bulb in this instrument is constructed of springs instead of the metallic arms hinged upon each other. This improvement, first made by Messrs. Meyer & Meltzer of London. has been adopted in this country by Messrs. Otto, and Messrs. Tieman & Co.

[†] Handbuch der systematischen Anatomie des Menschen von Dr. J. Henle, p 417.

disseminated by so many authorities, and which has achieved so much fictitious importance as a guide in urethral examination, viz., that the *meatus urinarius* is a *measure of the size of the normal canal.*[*]

What I desire now to make prominent, is the fact that the best recognized authorities have long appreciated the value of stricture as an agent in the prolongation of urethral inflammation and irritation. Whenever it could be demonstrated by the imperfect means used, it was at once accepted as the probable cause of trouble; it was only when no stricture could be found that the surgeon was driven to the use of internal medication and topical applications. The urethra was vainly explored for stricture, because the instruments in use were inefficient. The endoscope was the result of an intelligent effort to clear up the diagnosis in cases of gleet, where no stricture was found. Désormeaux, Cruise and others, discovered the granular spots studding the urethra in such cases, and the secret was apparently manifest. Topical applications through the endoscopic tubes apparently cured some, and gave temporary benefit to many; then an army of young endoscopists followed *en train,* believing, as taught, that the granular sensitive spots in such cases would, if not subjected to frequent occular inspection and intelligent cauterization, result in true organic stricture. And yet after months of faithful work in this direction, the return of gleet, without new contagion, made it evident that the true cause of gleet had not yet been reached in such cases. I have the record of at least a dozen instances * when the difficulty was proven to have been a stricture near the meatus, which nevertheless admitted the usual-sized endoscopic tube (22 or thereabouts), and the dependence of the granular spots upon this condition, proven by their complete disappearance upon the cure of the contraction, without the aid of any other treatment whatever; and this premises a conclusion arrived at by the experience gained in a very large number of cases, viz., that *gleet is always dependent upon stricture: that* while stricture may be present when no gleet is present; *whenever* there is a *gleet* in the sense of a chronic urethral oozing or discharge, an intelligent and thorough exploration, with suitable instruments, will *invariably* discover a *distinct* contraction of the meatus urinarius, or a *readily recognized* co-arctation of the uretha at some point; and further, that the complete restoration of the urethra to its normal calibre and suppleness at the contracted points will be required to warrant the statement that a *permanent cure* has been effected.

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[*] A constant relation appears to exist between the urethral calibre and the size of the penis with which it is associated. This is a fact demonstrated by careful measurements with the urethra-meter in several hundred cases, without exception being met. The proportion runs as follows: When the flaccid penis measures 3 inches in circumference, the size of the urethra will be 30 millimetres in circumference, or more. When it is 3¼ inches, it will be 32 or more; 3½ inches, 34; 3¾ inches, 36; 4 inches, 38; 4¼ to 4½ inches, 40 or more millimetres.—Where the urethra-meter is not available, this proportionate relation may be relied upon as not *overestimating* the *normal* urethral calibre in any case.

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* The following is the record of a typical case of this sort:

Mr. W., aged 25, came under my care December 1st, 1872. Contracted first gonorrhœa early in June 1872, was treated by injections locally, and alkalies internally, until August 1st, during which time he had no freedom from the discharge, nor from the acute suffering. At about this time, the vesical neck became involved, and he suffered most from frequent and painful micturition. Came under the care of Dr. ——, a skilled endoscopist, who discovered numerous granular patches in the course of the canal, extending quite into prostatic portion, and applications of a strong solution of nitrate of silver were made through the endoscope, which afforded temporary relief; urination still painful every hour. By September 1st, the discharge decreased to a slight mucus, following the use of pencils of tannin and glycerine. A spell of damp weather brought back the purulent discharge, with return of perineal pain and frequency of micturition. Tannin pencils again used, but after continuing for four weeks. and no improvement, patient was put to bed and hot hip baths were administered every two hours, etc., etc. After five weeks of various kinds of treatment, local and general, he came to me from his bed, December 1st, 1872. On examination I found no difficulty in introducing No. 20 F. bulbous sound and discovered a firm cartilaginous stricture from just within the meatus to half an inch back. This I freely cut with Civiale's bistouri cache. Immediately following the operation, he expressed himself as feeling " like a new man." The discharge ceased within twenty-four hours, the perineal pain and frequency of micturition, and the *ardor urinæ* also ceased, and he returned to his duties, which were most active, on the following day, after having been laid up for over five months. *The urethral granulations subsided, and finally disappeared within a few weeks without* any local or general treatment. His recovery was absolute and complete, and the only solution afforded was the *division of the stricture at the meatus*, to which the granular spots in the posterior part of the canal were undoubtedly due.

The complete division of stricture has, in my experience, resulted *uniformly* in its complete disappearance within a period varying from three months to one year, and the *cure of gleet has, as a rule, followed the complete division of stricture within a period varying from twenty-four hours to four weeks after the final operation.*

Let us now consider the various degrees in which gleet is found, presenting to the surgeon.

First. When it is just sufficient to form shreds of inspissated mucus, which are observed on examination of the first washings of the urethra during the act of urination.

Second. When it is in the form of a simple, transparent exudation, only sufficient to glue the lips of the meatus urinarius together, and not even enough to stain the patient's linen.

Third. When, on squeezing the penis and subjecting the meatus to pressure (as patients afflicted with gleet are very much in the habit of doing), a single drop of semi-opaque, or creamy purulent fluid may be made to ooze out.

Fourth. When it is met as a thin, profuse, nearly or quite painless discharge, easily reduced in amount by astringent injections, but as readily returning on their withdrawal, and, even if apparently cured, returning promptly on the least vinous or sexual indulgence.

Fifth. When the discharge, thicker, decidedly yellow, and persistently profuse, exudes from an inflamed and pouting meatus, usually causing much redness and irritation upon contact with the preputial tissues.

Each and all the grades or varieties of gleet above enumerated and casually described may, it is believed, be proven to owe their *per*sistence, if not their *ex*istence, to simple, localized, mechanical obstruction to the passage of urine.

The impetus which is given to this fluid during an ordinary micturition is of no insignificant character. The muscles of the diaphragm, abdomen and perinæum combine to bear down, press against, support, and steady the bladder, while the active agents, the detrusor muscles which interlace over the entire organ, exert a contractile force which is sufficient to overcome the resistance of the sphincter vesicæ, and project the urine in a full, smooth stream through the urethra, and to a distance of several feet. This, however, gives but a faint idea of the effect which a prolonged resistence to the power of the muscular apparatus concerned, in emptying the bladder, may produce. In order to be fully appreciated, this should be observed in a person laboring under some obstruction to the passage of urine, such as occurs in urethral stricture. If the stricture is a slight one it may be apparent only in producing a want of rhythm in the muscular action of the urethra, which prevents a prompt and complete emptying of the canal. Thus it is that *dribbling*, after the act, is occasioned. When the stricture encroaches to a somewhat greater degree, the stream is no longer full and strong, but becomes twisted, and is projected with less force, and now that the patient often finds himself exerting a pressure of many extra pounds in bearing down upon the bladder, the resistance of the stricture begins to be realized. But let the case be one where the stricture has closed the urethral lumen, so that a continuous stream is no longer possible. The pressure becomes so great, that, after a time, not only does the urethra become permanently enlarged behind the stricture, but the urine is pressed backward from the bladder through the ureters, resulting in dilatation of these delicate tubes to many times their normal size: the pelves of the kidneys also participating in this forced dilatation, until a positive sacculation may be produced. This power by which the urine is propelled, certainly furnishes the requisite conditions necessary to establish a point of irritation in a urethra when stricture is present. It is only necessary to establish the fact, that the *normal resiliency* of the urethra is diminished at a given point, to prove that, during micturition, a perturbation in the stream *must* occur at such point, even if it is not sufficient to attract attention in any way. Hence the slightest contractions assume an importance which could not be inferred from the *apparent* freedom from trouble in passing the urine. They establish a localized point of friction, and, of necessity, an increased excitement in the vessels of the part, possibly only enough to disturb the complete elaboration of epithelial material, and cause the shreddy deposit to take the place of the clear normal secretion; and this may occur with very slight, or not even the least abnormal sensation being present. The presence of the mucoid shreds in the urine may be the *only* evidence of commencing trouble. But a permanent point of friction once established, greater than the natural conservative power of the surrounding parts is able to counterbalance, obstruction is increased by the natural aggregation of plastic material at the point of irritation. In this way the tendency to recovery is combated, and a permanent point of inflammatory action is established.

Thus the difficulty, which commenced simply as an obstruction to the resiliency of the urethral walls, progresses naturally and certainly, to the point of narrowing to a greater or less degree the calibre of the urethral canal. The second point of importance is the incomplete emptying of the urethra after micturition, which occurs as a necessary consequence of anterior contractions. If the muscular structure is embarrassed, its function is imperfectly performed, and instead of completely emptying the canal of its irritating contents, a drop, or several, is retained, either to dribble away slowly within a few minutes after urination, or to be held behind the contraction by a spasmodic action (always readily set up in the vicinity of urethral irritations) until chemical changes heighten its irritative action, and it becomes capable of establishing new points of irritation, such as are seen in *granular urethritis*, so-called. It is not impossible nor improbable that, as Désormeaux and Cruise have taught, the granular spots found in the urethra in cases of gleet *may* be the beginning of stricture: but it is positively true that they may be, and most frequently are the legitimate progeny of an *already-formed stricture*, anterior to the point of their location, and it is equally true that unless stricture has already occurred as a result of the granular urethritis, the cure of the anterior co-arctation will result, without other treatment, in the disappearance of the granulations, and a complete restoration of the canal to its normal condition. The treatment of gleet by a systematic introduction of sounds and bougies, medicated or otherwise, is based upon the idea of a possible co-arctation of the urethra at some point. Ordinarily this plan is resorted to in the most empirical way, simply because the introduction of sounds and bougies is recommended by authorities for the cure of gleet. By our most intelligent surgeons, it is directed to the dilatation of strictures, which have been suspected, or detected by the bulbous sound or bougie, and with a full appreciation of the probable dependence of the gleet upon the presenting strictures.

That this plan, intelligently pursued, has often cured gleet, no one will for a moment gainsay; but that it permanently removes the cause, no one at this day is likely to affirm. Nothing is more distinctly laid down in the writings of authorities in regard to the treatment of urethral stricture, than that the results of dilatation are *always* of a *temporary* character. So that, it is well understood, in cases of the cure of gleet by dilatation of the stricture or strictures, upon which it is dependent, *subsequent* dilatation must be kept up *indefinitely*, at varying intervals, in order that the gleet may not again be established. For a permanent cure, a complete division of the contracting stricture must be had, and any treatment which falls short of this will, of necessity, fail in doing more than to temporarily remove the obstruction which has been the cause of the gleet. The radical cure of stricture was made the subject of a paper read by me before the New York State Medical Society in February last, and may be found in the Transactions of that society for the present year.* In that paper, the carefully tabulated results of 203 operations will substantiate, in some degree, the claim I have made in regard to the constant association of a stricture with gleet, and the results of operations as there recorded will also make it manifest that my confidence in the radical cure of stricture, as well as of the gleet which is so frequently associated with it, is not without reasonable foundation.

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* A reprint of this paper (as well as other papers on the same subject) is published by Putnam's Sons, cor. 23d St. and 4th av., New York.

APPENDIX.—STATISTICAL TABLES OF FIFTY CASES OF GLEET, OR CHRONIC URETHRAL DISCHARGE, ASSOCIATED WITH URETHRAL STRICTURE.*

No. of Case.	Age of Patient.	Number of Strictures.	Locality of Stricture.	Size of Stricture.	Norm. Calib. of Urethra.	Cause of Stricture.	Complications at Date of Operation.		Number of Operations.	Results and Remarks.
1	38	3	½ in.. 2 in.. 2½ in..	28 28 28	34	Gonorrhea fifteen years ago. Several times since. Last attack, four years since.	Pain in urethra, scrotum, thighs, knees, legs, feet, groins. Painful movements of testicles.	Gleet.	1	Immediate relief following operation. Recurrence of symptoms reported. No re-examination
2	32	1	Meat..	22	31	Gonorrhea ten yrs. ago.	....	....	1	Cure in six weeks.
3	68	2	Meat.. 2¼ in..	29 29	32	Gonorrhea 47 and 40 years previously.	Lumbar and perineal pain. Frequent micturition.	Gleet several years.	2	Immediate relief. Cure in one month.
4	54	1	1 in..	29	34	....	....	....	1	Cure complete in two weeks. Thirteen months after operation, no re contraction.
5	33	1	½ in..	..	..	Gonorrhea several times in last ten years.	....	5 years...	1	Cure complete in two weeks.
6	24	1	Meat..	20	32	Gonorrhea...........	Irritation in urethra.	Gleet.	1	Cure. One year after operation, no recontraction.
7	30	9	½ in.. 1 in.. 1½ in.. 2½ in.. 3 in..	23 28 28 30 30	37	Gonorrhea four years previous.	Frequent micturition.	Nearly 4 years.	3	Slight gleety discharge remaining ten days after the operation, not since heard from.

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* NOTE.—The accompanying cases are extracts from the statistical records of one hundred cases of Urethral Strictures reported to the State Medical Society of New York in a paper on the "*Radical Cure of Stricture*," and published in the transactions of the Society for 1875. The cases were observed, more especially, in the view of their connection with Urethral Stricture. The accompanying Gleet was an incidental matter, and the exact dates of recurrence and duration were not as carefully noted as they should have been. It will be safe, however, to assume that in no case did THE Gleet exist, as a persistent or recurring discharge, less than several months, before the operative procedure which resulted in its cure.—F. N. O.

			3¼ in.. 4 in.. 4½ in.. 5½ in..	30 28 28 28						
8	45	1	1¼ in..	30	32	Gonorrhea 15 yrs. ago. Several times since.	....	Gleet 12½ years.	1	Cure in one month.
9	..	1	Meat..	21	32	Gonorrhea four months previous.	....	Gleet 3 months.	1	Cure.
10	25	1	½ in..	20	34	Gonorrhea six months previous.	Pain in perineum. Frequent and painful micturition.	Gleet.	5	Cure. Four re-contractions with partial return of symptoms. Final cure after last operation.
11	25	2	1⅓ in.. 6 in..	23 21	31	Gonorrhea three years previous.	....	1 year....	1	Cure. Deep stricture not divided.
12	25	4	2 in.. 4½ in.. 4¾ in.. 5 in..	24 24 24 24	30	Gonorrhea one and a half and one year previous.	....	Gleet 10 months	2	Cure. Two and three years after operation no re-contraction.
13	20	2	⅓ in.. 1½ in..	24 24	30	Gonorrhea and masturbation.	....	Gleet.	2	Cure. One year after operation, no re-contraction.
14	30	3	¾ in.. 2½ in.. 3½ in..	..	..	Gonorrhea ten years previous.	....	Gleet.	2	Cure.
15	50	1	⅛ in..	18	29	Gonorrhea 30 and 25 years previous.	Painful and frequent micturition.	Gleet.	2	Cure. Re-contraction after six months. Second operation. Relief which remains permanent.
16	54	2	⅛ in.. 3 in..	16 26	31	Gonorrhea..........	Irritability of Vesical neck.	Gleet.	2	Cure. Remains complete three years after last operation.
17	35	4	Meat.. ½ in.. 1 in.. 2 in..	19 19 27 27	31	Masturbation........	....	Gleet.	2	Cure of Discharge.

STATISTICAL TABLES—*Continued.*

No. of Case.	Age of Patient.	Number of Strictures.	Locality of Stricture.	Size of Stricture.	Norm. Calib. of Urethra.	Cause of Stricture.	Complications at Date of Operation.		Number of Operations.	Results and Remarks.
18	40	1	½ in..	36	38	Gonorrhea three years previous.	Frequent micturition Sense of foreign body just behind meatus, causing great nervousness.	Recurring Gleet.	1	Cure within two weeks.
19	19	7	1 in.. 2¼ in.. 3 in.. 3½ in.. 3¾ in.. 4 in.. 5½ in..	25 31 26 22 31 31 27	31	Gonorrhea two years previous.	Frequent and painful micturition. Repeated urethral chills, caused by attempted dilatation.	Gleet many mos.	1	Cure.
20	24	2	Meat.. 2 in..	29 29	31	Gonorrhea..........	Spasmodic stricture at seven inches.	Gleet several years.	..	Immediate relief of spasmodic stricture. Prost. abscess reported ten days after.
21	25	1	½ in..	22	32	Gonorrhea six years ago. Frequently since.	....	4 years...	1	Cure.
22	29	5	⅓ in.. 3 in.. 3¼ in.. 3½ in.. 4½ in..	24 25 24 26 29	..	Gonorrhea six months previous.	Cystitis. Vesical tenesmus. Pleurisy with effusion. Aggravation of symptoms from diuretics.	6 months.	1	Immediate relief of all symptoms connected with the urinary organs. Tolerance of diuretics re-established.
23	38	2	..... 2 in..	26 30	32	Gonorrhea six years previous.	Painful micturition. Spasmodic stricture.	Recurring Gleet.	1	Cure of troubles within a month.

24	41	2	Meat.. 1 in..	24 24	30	Gonorrhea six years previous.	Unpleasant sensation in testicles.	Gleet....	1	Cure.
25	28	5	Meat.. 1 in.. 1¾ in.. 2 in.. 2¾ in..	22 31 31 31 31	34	Gonorrhea six and five years previous.	Frequent and painful micturition. Weakness.	Gleet....	2	Cure of gleet and painful micturition.
26	28	1	¼ in..	30	34	Gonorrhea seven years ago.	....	Gleet...	2	Cure for one month, when patient acquired fresh gonorrhea.
27	29	5	Meat.. 2¼ in. 3¼ in.. 4½ in . Memb Port'n.	9 9 9 9 8	32	Gonorrhea...........	Frequent and painful micturition. Frequent attacks of retention of urine.	....	1	Recovery with 30f. calibre.
28	21	5	Meat.. ¼ in.. 2 in.. 2¾ in.. 1 in..	26 26 26 26 30	36	Gonorrhea three and a half years and two months previous.	....	Gleet...	1	Cure of gleet within two weeks.
29	28	7	Meat.. 1 in.. 2 in.. 2¼ in.. 1½ in.. 4 in.. 4½ in..	22 22 30 30 30 30 30	32	Gonorrhea six years previous.	....	6 yrs. Gleet	4	Cure.
30	25	5	¾ in.. 1¼ in.. 1½ in.. 3 in.. 4 in..	20 20 20 27 27	28	Gonorrhea ten and seven years previous.	... .	7 yrs. Gleet	3	Cure.

STATISTICAL TABLES—*Continued.*

No. of Case.	Age of Patient.	Number of Strictures.	Locality of Stricture.	Size of Stricture.	Norm. Calib. of Urethra.	Cause of Stricture.	Complications at Date of Operation.		Number of Operations.	Results and Remarks.
31	..	4	Meat.. 2¼ in.. 2½ in.. 3 in..	23 26 30 31	32	Gonorrhea five years previous.	....	Gleet....	4	Cure.
32	..	1	Meat..	22	32	Gonorrhea twelve years ago.	Frequent erections. Urinary sinus near meatus.	Gleet 1 yr.	3	Cure. Sinus healed.
33	25	5	1½ in.. 3 bnds. 4½ in.. 2 bnds.	20 27	30	Gonorrhea ten years previous.	Irritable bladder.	Gleet recurrent.	2	Cure.
34	..	8	Meat.. 2¼ in.. 2½ in.. 2¾ in.. 3 in.. 3¼ in.. 4½ in.. 5 in..	22 22 26 24 30 30 24 24	31	Gonorrhea............	....	Gleet....	5	Cure.
35	..	3	2¾ in.. 2¼ in.. 3¼ in..	29 27 27	30	Gonorrhea two and one and a half years previous.	....	Gleet....	5	Cure.

36		1	$\frac{3}{8}$ in..	24	30	Gonorrhea five and two years.		Gleet....	2	Cure.
37	23	10	Meat..	24	40	Gonorrhea one year previous.		Gleet....	8	Cure.
			$\frac{3}{4}$ in..	22						
			1 in..	31						
			$1\frac{1}{4}$ in..	22	..			$2\frac{1}{4}$ years..		
			$1\frac{1}{2}$ in..	31						
			$1\frac{3}{4}$ in..	31						
			2 in..	33						
			$2\frac{1}{2}$ in..	37						
			$3\frac{1}{2}$ in..	37						
			$4\frac{1}{2}$ in..	37						
38	27	1	Meat..	24	30	Gonorrhea..........	Spasmodic stricture at membraneous portion.	Gleet....	1	Cure.
39	32	1	Meat..	22	30	Gonorrhea twelve years previous. Masturbation.	Frequent seminal emissions. Nervousness.	Gleet....	1	Cure of Gleet in two weeks. Married at end of one month. Re-examined two months after. No return of trouble.
40	40	1	$\frac{1}{3}$ in..	26	30	Gonorrhea 12 years previous.	Frequent micturition. Pain in penis. Intense pain following seminal emissions.	Gleet 14 months.	2	Cure. Relief of pain, and frequent micturition. Cessation of discharge, and frequent micturition for three months, when they returned. Recontraction found. Second operation followed by renewed relief, which continued six months, when he contracted a fresh gonorrhea.
41	50	1	$\frac{1}{2}$ in..	18	30	Gonorrhea twenty-five years previous.	Irritability of bladder.	5 yrs. Gleet	2	Cure. Return of symptoms five months after first operation. Re-examined two and a half years after second operation. Has remained perfectly well.
42	35	1	$\frac{1}{2}$ in..	25	34	Gonorrhea..........		Gon. acute for 5 mos.	2	Immediate relief to acute symptoms.

STATISTICAL TABLES—*Continued.*

No. of Case.	Age of Patient.	Number of Strictures.	Locality of Stricture.	Size of Stricture.	Norm. Calib. of Urethra.	Cause of Stricture.	Complications at Date of Operation.		Number of Operations.	Results and Remarks.
43	32	2	Meat.. 3 in..	34 19	38	Gonorrhea seven years previous. Several times since.	....	Gleet.... Recurring Gleet.	2	Relief of discharge.
44	29	2	$\frac{1}{3}$ in.. $2\frac{1}{4}$ in..	26 29	30	Gonorrhea three months previous.	Spasmodic stricture.	Gleet....	3	Cure of gleet. Relief of spasmodic stricture.
45	28	4	$1\frac{1}{2}$ in.. $2\frac{1}{8}$ in.. $2\frac{1}{4}$ in.. $2\frac{5}{8}$ in..	16 21 21 21	31	Paraphimosis. Accidental.	Retention of urine.	Gleet 6 months.	3	Relief of symptoms.
46	40	1	Meat..	28	32	Gonorrhea seven years previous.	Difficult micturition, followed by pain in urethra.	Gleet....	1	Cure.
47	29	1	$\frac{1}{6}$ in..	26	31	Gonorrhea 2 years, also 2 months previous.	Burning in urethra during micturition. Pain in back.	Gleet ...	1	Cure.
48	54	3	$\frac{3}{4}$ in.. $1\frac{1}{4}$ in.. $2\frac{1}{4}$ in..	20 24 28	31	Gonorrhea twenty, also eight years previous.	Frequent and painful micturition. Pain in penis, testicles perineum, thighs. Attacks of retention of urine. Chron. Cystitis.	Gleet several years recurrent.	2	Cure. One month after first operation. Re-contraction. Redivision of stricture at meatus. Relief. Perfectly well three months after, as reported by his physician.
49	..	1	Meat..	16	30	Gonorrhea	....	Gleet....	2	Cure.
50	47	1	$\frac{1}{2}$ in..	22	28	Gonorrhea twenty, also three years previous.	Return of gleet after each venereal indulgence.	....	1	Cure.

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ON THE DIAGNOSIS OF DISEASES ACCOMPANIED WITH REAL OR APPARENT PARAPLEGIA WITHOUT MARKED MUSCULAR DEGENERATION.

BY

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GENTLEMEN: You know too well to need any telling what is meant by paraplegia, but perhaps it may not be wasting time for me to say in what sense apparent paraplegia is used in the announcement of my subject. Apparent paraplegia may arise from suppression of movements by various local diseases of the legs. These I only mention to caution you to closely examine patients, else some time you may suddenly wake up to the fact that you have been treating as nervous, a paralysis due to an inflamed joint, muscle, or bursa. The apparent paraplegias I intend to discuss to-day, are such as accompany spinal affections, like loco-motor ataxia—where there is no local disease and no real paralysis, although the patient is unable to walk. Perhaps, indeed, a better title for the present discourse would be the diagnosis of diseases of the spinal cord, but I have preferred the one chosen as fixing the attention upon one chief symptom, and because, for want of time, I will be forced to leave out of sight one class of spinal affections. Before taking up the subject immediately in hand, allow me to say a few words in regard to paraplegias of cerebral origin. But very few cases of this character have been reported, and in the majority of these the spinal cord was not examined at the autopsy, so that it is very possible that a spinal lesion co-existed with that of the brain. Still it has happened, and may happen again, that a double cerebral lesion, or a single lesion in the pons varollii shall produce a paraplegia. The diagnosis is to be made out by the existence and relations of distinct evidences of brain diseases, in the form of headache, impaired cerebration, and paralysis of parts supplied by nerves arising above the spinal cord. Individual cases will occasionally present themselves in which you will find it for a time very difficult to decide whether the paralysis be of cerebral or spinal origin. But a few days ago, I saw a case in consultation, in which I was forced to confess my inability to positively determine the point at once. The only course under these circumstances is to wait.

The subject of spinal diseases is, by students, usually considered very difficult, yet it is really simple, if only too great pathological refinements are avoided. Of acute disorders of this class there are but very few, and most of the chronic affections are resolvable into various degenerations, which may well be grouped together as chronic myelitis. It is a very vital point to remember that loco-motor ataxia and other spinal organic disorders are not, pathologically speaking, diverse entities; they are rather clinical groups of cases which may shade into one another. One pathological process giving origin to various of these so-called diseases, according as it is located in various regions of the spinal cord, and giving rise also to hybrid affections, by appearing simultaneously in various localities.

The symptoms of organic spinal disease, are produced by the excitation, the depression, or the total abolition of the functional activity of the cord. It is plain, that in order to understand these symptoms, it is necessary to know the physiology of the spinal marrow. The chief functions of the cord are: to give passage to peripheral impulses from without up to the brain; to return from the brain impulses to the voluntary muscles ; to receive the peripheral impulses, and return them directly to the muscles so as to produce reflex movements ; to, as I believe, bind the voluntary muscles together so that they may be coördinated ; to aid in vaso-motor control, and to affect more directly the nutritive processes of the body.

Knowing the general functions of the spinal marrow, it is next necessary to study the various seats of these functions.

For clinical purposes, the cord may be divided into three regions, the posterior columns, the gray matter, and the anterior or antero-lateral columns. The antero-lateral columns have for their chief functions the conveyance of motor impulses from the brain downwards, or from the reflex centres outwards, so that their section is followed by complete arrest of voluntary movement. Much more complex physiological powers are possessed by the posterior columns, which are chiefly composed of prolongations of the posterior nerve roots, passing through to the gray matter of the cord, and of longitudinal fibres, which go from one portion of the gray matter to another. These fibres are probably commissural, uniting together the motor cells of the cord. It is probably to them that the posterior columns owe the control which they certainly exercise upon coördination, or the power of regulating the movements of large groups of muscles to accomplish a given end. It is very hard to understand how such a distant nerve mass as the cerebellum can so control groups of muscles as to regulate their action ; it is very easy to perceive how commissural fibres shall so bind even distant nerve-cells, one to the other, that they shall act in complete unison, even indeed, as a single centre. Although I do not think it has been proven beyond cavil, yet it is probable that the cerebellum has a coördinating function, but the fact that pigeons deprived of that organ recover their coördinating power, as they recover from the injury, proves conclusively that the cerebellum is not the *only* organ of coördination. Destruction, by disease, of the posterior columns of the cord certainly leads to loss of coördination, and, I think, it is extremely probable, that these tracts of white nerve-matter control muscular movements through their longitudinal commissural fibres, whilst through their horizontal fibres, the prolongation of the sensory nerve roots, they certainly transmit sensory impulses to the gray matter. If, by disease, the functional activity of these posterior columns is lowered, anæsthesia and loss of coördination must result, but if there is simple excitement of the part, pain, with more or less marked hyperæsthesia must be produced. Usually, in disease of these columns, there is excitement and irritation, along with depression of function. Thus, if an exudation presses upon the nerve tubules in the posterior columns, there must result from the interference with the horizontal sensory root fibres, intense pain, and partial or complete anæsthesia, and from disturbance of the commissural fibres more or less loss of coördination.

The gray matter of the cord is insensitive, and, therefore, its degenerations are not productive of much pain. Through it are transmitted the sensory impulses, and therefore its destruction produces anæsthesia. It is the seat of the reflex centres, and, consequently, when it is excited by disease, the reflex movements are over-active. In its anterior cornua are situated certain cells, which appear to be trophic in function, and when these are destroyed muscles degenerate.

Outside of the cord are the membranes. The outer two of these are the chief. They are separated from the cord by the, comparatively speaking, wide subarachnoid space. In inflammation of the spinal meninges usually these two membranes are chiefly concerned, and, owing to the space between them and the cord, the latter does not participate very profoundly in the disturbance. It is very different with the nerve-roots. These do not pass directly through openings in the spinal envelopes, but are for some distance closely bound around with sheaths formed of prolongations of the membranes. Owing to this close union, when the membranes are inflamed the nerve-roots feel much more than the cord the effects of the disorder, and soon come to share in the inflammation. Frightful pains, and no less frightful spasms, darting and pirouetting through the limbs, mark the excitement, not of the membranes, nor of the cord, but of the spinal nerve-roots. No paralysis can appear until sufficient exudation to abolish nerve-function by pressure has taken place, or until the neuritis has so advanced as to destroy the continuity of the nerve structure. In chronic, as well as in acute, spinal meningitis, very rarely, if ever, is the exudation, or the intensity of the neuritis, sufficient to completely abolish function, and, consequently, pain and spasm predominate over paralysis even in the advanced stages of the disease. The sensory nerves are so much more easily disturbed in their functions than the motor that, with the pain of meningeal inflammation, there is usually some degree of anæsthesia; but paralysis of motion is not a symptom of the disease. The patient, paraplegic from such cause, lies in bed not with the soft, extended, flabby limbs of muscular paralysis, but with his legs tightly flexed upon the body, the muscles hard, and the tendons tense from the unyielding spasm. When the meningitis is partial or is very mild, of course the spasms are partial or very slight.

In approaching any case of disease of the spinal cord proper, it is always necessary to determine first, whether it be organic or functional. The following classification of diseases giving rise to paraplegia, will serve to guide us in our study of these diseases:

Organic.	*Functional.*	*Hysterical.*
Disease of cord.	Anæmic.	Hysteria.
	Reflex.	
	Dyscrasic.	

Before going further it is right that I should explain to you very briefly it must be, what is meant by anemic, reflex, and dyscrasic paraplegia. If you bleed an animal frequently and largely, not rarely there results a partial paralysis of spinal origin, so that the hind-legs are dragged in walking. Precisely similarly to this, in man anæmia, from true or from serous hemorrhage, may produce a partial, or, perhaps, even a complete paraplegia. It is foreign from the purpose in hand, to inquire as to the mode in which the paraplegia is induced. But here is a patient from my ward in the Philadelphia Hospital, offering an example of this variety of paraplegia. He tells us that in July he was seized with a furious cholera morbus; serous stools for forty-eight hours being incessant. At first they were very large, but, afterwards, as the body was drained of its liquid, they became smaller. As the purging ceased he noticed that he was losing power in his legs, and this paralysis increased rapidly for a day or two until he was unable to walk at all, then became stationary, and after a time, under treatment, commenced slowly to amend. I want to call your attention to the torpidity, so to speak, of the symptoms (at no time has there been any pain, save some numbness and prickling); to the fact that both motion and sensation are involved, but have neither of them been completely abolished; to the circumstance that the bladder has remained free, and to the evident cause of the trouble. This I take to be a typical case of anemic paralysis, and, as you see, the symptoms closely resemble those of mild congestion of the cord. The diagnosis is impossible, between the two affections, without the history.

In some way or other, not positively determined, an intense peripheral irritation may produce a palsy. This is especially seen in connection with an irritation of the genital tract; in man, often in affections of the kidneys, or of the bladder, especially in stone; in boys from adherent elongated prepuce; and in women from uterine irritation, as that of a tumor. It is paralyses of such character that are classed as reflex.

Under the head of dyscrasic paraplegias, I refer to the palsies which sometimes follow diphtheria and other similar constitutional affections.

The term functional, which I have employed to designate this group, might be objected to ; but do not let us waste time on this. The group is, clinically, a very useful one, and the name is a matter of little importance. Hysterical paraplegia is undoubtedly functional, but I have separated it because, in its diagnostic peculiarities, it resembles more closely the organic palsies than those of Group 2.

The important question here naturally arises : Are we able to determine, from intrinsic characters, to which of these groups a given case belongs? Usually, gentlemen, we are, but not always. In the so-called functional paraplegias the onset is always more or less gradual. The disease may sometimes develop rapidly in the course of a day or two, but, as far as I know, never abruptly. In the organic the attack may be almost instantaneous or very rapid, but probably it is more often gradual. In the hysterical variety the paralysis is generally developed at once. In the organic paraplegias you usually have, at some period of the disease, indications of a motor or sensory functional excitement in the form of spasm or of pain in the affected limbs. In both the hysterical and the functional varieties you rarely, if ever, have any such indications. There is one especial form of pain, very possibly connected with spasm, which appears to be actually diagnostic of organic diseases of the spinal cord or membranes. It is the sensation of a band or cincture around the waist. I have seen partial anæsthesia in cases which I believed to be functional ; but complete abolition of sensation, very rarely, if ever, occurs. In hysterical paraplegia it is probably more common. There is one modification of sensibility which appears to rank with the cincture in its diagnostic importance. This is the retardation of the passage of a sensation from the periphery to the centre, so that a very perceptible time elapses between the patient's seeing his feet touch the floor and feeling that they do so. I think you may look upon this as decisive in any case.

Excitation of the reflex activity is, if present, an almost decisive proof of the existence of organic spinal disease. In hysterical paraplegia the bladder may become involved ; but in a patient suffering from a spinal affection, not hysterical, if difficulty be experienced in passing water without the presence of some obstruction, or if incontinence and a tendency to retention supervene—that is, if symptoms of paralysis of the bladder are developed—you may know that your patient has an organic spinal affection.

But suppose in any case you found that the onset had been gradual ; that there had been no pain or spasms ; that sensibility was not disturbed ; that reflex movements were not and had not been increased ; that the bladder was unscathed ; would it be certain that the patient was not suffering from an organic spinal affection ? No, gentlemen. In such a case as that which I have imagined, it is necessary to go over the various possible causes of paraplegia *seriatim.* Search if there be a stone in the kidney or bladder ; or, in a woman, if there be uterine tumor to start the chain of phenomena ending in reflex paraplegia. See if there exist any dyscrasia, or, if anæmia be present ; look whether the sufferer be hysterical. Very rarely, however, will this diagnosis by exclusion be necessary in cases of organic spinal disease, except it be in the very earliest stages of the disorder. Attention to the points already detailed, will almost always enable you to arrive at a rapid and correct conclusion.

The diagnosis of hysterical paraplegia may give you some trouble, as it at times very closely resembles organic disease. If distinct symptoms of hysteria are present or have existed in the past, very generally hysteria is the cause of the present paralysis. But organic paraplegia may occur in a woman suffering from hysteria, so care must be exercised not to mistake the relation of co-existence for that of causation. If you can learn, that under the influence of strong mental emotion, or from any other cause, the paraplegia has at times suddenly ceased temporarily, you may be very positive in your conclusion. It has been stated that in all cases of hysterical palsy, electro-sensibility is lost, although electro-contractility may be normal. This is certainly not correct. I have a number of times seen the electro-sensibility normal in cases of palsy, whose hysterical nature was undoubted. If, however, you find in any case that when you apply a strong faradic current, it is not comp'ained of, although the muscles contract strongly, and although general sensibility is preserved, you have very good reason for setting down hysteria as the *fons et origo mali.*

In a patient suffering from organic spinal disease, it is of the utmost importance to decide whether the disease be primary or secondary to an affection of the bony envelopes. I have at present under my care, a case which was presented to me as one of brain disease, probably tumor, because there was nearly complete paralysis of motion on the right and of sensation on the left side, with a history of very severe paroxysmal headaches. My attention was at once arrested by the absence of facial paralysis, and close inquiry soon showed that the headaches had existed for many years and were evidently migraine. I also found that the cerebration was perfect. The patient complained bitterly of great pain constantly in the right suprascapular region, and shooting up the neck and across to the other shoulder. This pain, the man stated, was greatly increased by a jar, so that he was unable to sit in any vehicle in motion, but was forced to stand, even in a street car, so as to lessen the effect of the jar. In attempting to put atropia in the eye of the patient, I found that he was totally unable to throw his head back, and some spasm of the muscles was apparent; deep lateral pressure upon the upper cervical vertebræ caused decided pain, as did also forcible downward pressure on the head or attempting to bend the neck. Finally, Rosenthal's test was applied, and the response of caries was obtained. The man had a history of syphilis, and the diagnosis was at once made of specific disease of the vertebræ causing a localized spinal affection; a diagnosis, I may mention, which the subsequent progress of the case has confirmed. The reason of the cross paralysis was very plain; but one lateral half of the cord was compromitted, and as the motor conductors of the cord decussate in the medulla, whilst the sensory conductors cross in the spinal marrow itself, the motor nerves of the side of the lesion were involved, whilst the sensory nerves of the other side suffered.

As some of you may not be familiar with Rosenthal's test, I may mention that it consists in passing down the back a pair of electrodes attached to a faradaic battery of some power, one pole being placed upon each side of the spine. Under these circumstances, if there be any caries or inflammation of the vertebræ, the moment its locality is reached the patient starts or screams from the burning, sticking pain caused by the passage of the galvanic current through the inflamed tissue. I have not found this test as trustworthy as its originator claimed it to be, and as, apparently, it ought to be. I have seen the local pain produced when the bony canal was not the seat of the disease. In cases simulating caries, however, the pain is probably not so severe as where the vertebræ are really affected. Moreover, absence of the pain in any case seems to be conclusive evidence of the non-existence of bone-disease. The nature of the cases which responded to Rosenthal's test, although there was no spinal caries, remained obscure, but there was some reason for believing that there was vertebral periostitis.

Having determined that neither the bony nor other envelopes of the cord are affected in a case of spinal disease, you should notice whether there have been marked trophic changes or degenerations of the muscles. I do not here refer to the ulceration and sloughing seen in myelitis, but rather to a rapid loss of galvanic irritability, and to wasting of the muscles without sloughing, such as occur in infantile palsy, and in progressive muscular atrophy. Passing by these affections, the diseases of the spinal marrow may be classified according to the rapidity of the onset, the attack being considered rapid when decided paralysis has developed within forty-eight hours as follows :

Rapid onset.	*Slow onset.*
Congestion.	Sexual exhaustion.
Spinal apoplexy.	White softening.
Meningeal "	Chronic myelitis.
Acute myelitis.	Tumors.

Acute Affections of the Spinal Cord with rapid Onset. In acute *congestion of the cord*, the large plexus of blood-vessels which surround the spinal marrow become intensely congested, and, in many instances, serous exudation aids in the production of the paralysis. The symptoms in spinal congestion being due solely to pressure, are, of course, simply those of depression of function, loss—of voluntary motion, reflex activity, and sensation. There are no spasms and no pain except that of tingling and numbness. The paralysis of motion is always more marked than that of sensation, probably for two reasons. As pointed out by Ollivier, the anterior portion of the cord is kept close to the posterior face of the vertebræ by the spinal roots, whilst the posterior portion is five or six lines from the corresponding face of the canal. A probably more efficient cause is the circumstance, that sensation is transmitted by the central deep portions of the cord and the motor impulse by the superficial layers, which, of course, feel first the effects of external pressure. Whatever may be the reason of the difference, the clinical fact is well established.

To recapitulate. In congestion of the cord the diagnosis rests upon : Suddenness of onset, uniform, bilateral loss of voluntary motion, reflex activity and sensation ; absence of all symptoms of irritation, such as spasms or violent pains; absence of constitutional disturbance. It must also be remembered, that the palsy affects first and most severely the lower limbs but may rise to the arms, and, finally, to the muscles of respiration and thus prove fatal; that so far as the paralysis extends, all the muscles are involved; that motion is affected more than sensation; and that very rarely, if ever, does ulceration or other indications of trophic changes occur. In *meningeal apoplexy* the symptoms are also due to pressure, but the effused blood not only disturbs the cord by pressing upon it, but also irritates the membranes and the nerve-roots, especially when first thrown out. Consequently, in the first few hours or days of a meningeal hemorrhage, there are violent spasms and pains, due either to an incipient meningitis, or more probably to a direct irritation of the nerve-roots. The extent and amount of the symptoms vary, of course, with the position and amount of the hemorrhage. Here, on the table, is the cord from a man who died recently in these wards; you see the enormous clots which everywhere abound below the upper dorsal region of the cord, but are especially large and abundant about the cauda equina. They everywhere pressed upon the lower cord and its nerves, as to totally abolish every motor, sensory, and trophic function. Let me read you, from the hospital Clinical Record, the history of the case. I may first state, that the cause of the hemorrhage was, in all probability, atheroma of the spinal vessels.

"John Huston, a teamster, aged 54 years, entered April 17, 1875. He stated that he had never suffered from any venereal disease, and had enjoyed good health until September, 1874, when he was injured in the side by a runaway team. He had just recovered from this, when he was seized with a violent pain in the ankle. During the winter of 1874–75, he was confined to bed with the 'rheumatism,' which began in the manner just noted. There were violent pains, swellings of the joints, and great soreness of the joints and muscles. He was not able to go to work before the latter part of March. He drove a team for four days a week for two weeks. April 8th, he went to bed feeling well, but was awakened at one A. M. with violent 'cramp pains' in the legs, soon spreading to the abdomen. Walking relieved the suffering, but the moment he laid down the pains would recur, accompanied with violent muscular contractions. So he spent the night, walking up and down the room. Towards morning the pain was gradually replaced by numbness and tingling in his 'legs, as though the crazy bone had been struck.'

"During April 9th he was able to walk, although he could not control his legs, which very frequently jerked about in all directions. At night the legs were very numb, though free from pain.

"The next day (10th) he was unable to walk, and on the 11th he began to lose control over his bladder. From this time until his admission into the house, no new symptoms were developed.

"April 17th.—His legs are pale, large, flabby, and his feet slightly œdematous. On the under lower edge of each foot, in a position where there can be no pressure during lying, is an unhealthy bulla, full of a clear sanious fluid. There is complete loss of voluntary motion below the knee in both legs; the muscles of the thighs he can contract slightly. Reflex movements are almost abolished in right, and very much impaired in the left leg. The sensibility is greatly diminished. On the right leg he cannot distinguish the compass points at less than six and one-half inches; on the left leg at less than ten inches. The electro-sensibility is very much diminished; the contractility (faradaic) abolished in right leg, except in tibialis anticus; in the left leg very greatly reduced. He suffers from urinary retention, with incontinence, the bladder being almost completely paralyzed. The urine, drawn off twice a day with a catheter, reddish-brown, acid, free from albumen. Bowels only opened by medicines; fæces and urine passed unconsciously. There is no loss of sensation in the arms, but there is some loss of power in the right arm. The radial arteries are exceedingly atheromatous, rigid as pipe-stems; temperature not elevated.

"April 23.—Since the last note the symptoms have steadily increased in severity, the bullæ on the feet have become gangrenous, and a general typhoid condition has developed. On the 21st, the respiration commenced to be affected, and is now shallow and labored.

"April 26.—Man dead.

"Autopsy.—Substance of the cord normal. The whole spinal canal as high as the middle dorsal region, filled with clots, which are especially abundant, large, and firm in the region of the cauda equina."

The relation of the symptoms to the lesion are so evident in this case, that I will not occupy time in commenting upon them.

A case which I recently saw in the Episcopal Hospital, under the care of Dr. Wharton Sinkler, will serve to illustrate the mild form of meningeal hemorrhage. A man went to bed feeling well, waked up in the morning unable to stand, and suffered from pain and spasms, with nearly complete loss of sensation and motion for some days. Then the pain and spasm disappeared, sensibility commenced to return, reflex actions were greater than normal, but there were no spasms. If the spinal cord be divided by the knife of the vivisector in animals, or of the assassin in man, so low down as not to interfere with respiration, the reflex movements are always greatly exaggerated below the point of division, probably on account of the removal of the influence of an inhibitory centre at the base of the brain, the so-called Setschenow's Centre. The persistent increased reflex activity in the case of which I have been just speaking, proved that the clot was a local one.

Without further discussion it may be asserted, that the diagnosis of *meningeal hemorrhage* depends upon the extreme suddenness of the onset; the first symptoms of irritation, pain, and spasm ; the after occurrence of symptoms of pressure, varying in intensity with the amount of the effusion ; the absence of febrile disturbance unless decided meningitis be produced by the clot.

In *spinal apoplexy* the symptoms come on with absolute abruptness. The cord is so small a body that a clot in its substance interrupts at once its function. The paralyses of motion and sensation are complete, and reflex movements are greatly exaggerated. As there is no correlation of the spinal nerve-roots, the spasms and pains of meningeal hemorrhage are wanting.

At first sight the diagnosis appears to be a most easy and certain one. The extreme suddenness of the attack, the completeness and abruptness as to limit of the palsy of motion and sensation, the marked exaggeration of the reflex movements and the absence of the symptoms of meningeal irritation, pain and spasm, make a picture not readily mistaken. A clot outside of the membranes of sufficient size to press upon the cord severely, may, however, induce exactly the symptoms just detailed, and it is doubtful whether the diagnosis between the two affections is possible. In the only case of hemorrhage external to the spinal membranes, which I have seen, there was a zone of over two inches in width, between the line of perfect sensation and that of complete loss. In spinal apoplexy the boundary of the anæsthesia ought to be more abrupt, and, it may be, that the difference is of diagnostic value.

Acute primary myelitis is a very rare affection. The diagnosis should present no difficulty. The distinct febrile reaction, which is stated to be always present, separates it at once from all other acute affections of the cord proper, so that it can be confounded only with acute meningitis. Probably, in the majority of cases, it exists coincidently with the last disorder ; but even when it is isolated the symptoms at first closely simulate those of meningitis. There are the same pains and spasms in the membranes, but the pains are less severe, and are less general, and are, before long, accompanied by marked loss of sensibility, passing into a complete anæsthesia dolorosa. The convulsive movements are also less severe than in meningitis and far less persistent, being in a very few days replaced by paralysis. The reflex movements at first exaggerated, now disappear, and local death, ulceration and sloughing, betray the grave affection of the trophic centres. The diagnosis, at first difficult and, perhaps, of necessity uncertain, now becomes but too evident.

Before taking up the more usual forms of chronic spinal diseases allow me to call your attention to *spinal tumors*, including in this term every morbid growth or process that produces a gradual compression of the spinal cord. Sufficient was said early in the lecture in regard to Potts' disease, but let me call your attention to the fact that it was shown that there are three classes of phenomena to be looked for in this disease: local symptoms of diseased structures; atrocious pains at a distance from the seat of the disease, due to the involvement of nerve-roots and nerves, where they pass through the inflamed tissues; and paralytic symptoms, the results of pressure, and to some extent of a local myelitis. In cases of suspected tumors of the spine, all these symptoms are to be looked for. In cancer they are often all present, and the distant pains are especially remarkable for their atrocity. The symptoms of pressure are, of course, paralysis of motion and sensation gradually deepening. This may be symmetrical, but very often it is not so, and when it takes the hemiplegic form the anæsthesia and loss of motility are upon opposite sides of the body. In tumors developed within the vertebral canal the pain of nerve involvement is often wanting, and the diagnosis must be made by the process of exclusion. The existence of paralysis of motion on one side of the body, and of sensation on the other, shows that there is a local destruction of the function of a lateral half of the cord; a condition which, when gradually developed, is always produced by pressure from without. It must be remembered that the reflex actions in the paralyzed part are above normal, because the lower section of the spine is removed from the controlling influence of the cerebral centres. In some cases there are very violent reflex spasms, both clonic and tetanic, producing certain symptoms which I shall speak of more in detail under the heading of local myelitis. At times the existence of multiple tumors outside of the spinal cord will aid you in arriving at a diagnosis. Thus in a case of leukæmia, not long since in the Pennsylvania Hospital, a correct diagnosis of the cause of the existent paraplegia was arrived at because of the general lymphatic hyperplasia. Sometimes the secondary affections of the cord will have so advanced before the case comes under your eye as to make the diagnosis impossible.

Leaving out of sight tumors, chronic spinal diseases may be divided into the following classes :

Without tremors.	*With tremors.*
Sexual exhaustion.	Paralysis agitans.
White softening.	Multiple sclerosis.
Chronic myelitis { softening, sclerotic.	
Local myelitis.	

Sexual exhaustion might be considered a functional disorder if indeed it were really worthy of being recognized as a distinct disease. The difference between it and myelitis is probably only one of degree. Still, there seems to me a practical clinical advantage in the use of the term. A man abuses his sexual powers, suffers from paraplegia, comes under treatment and gets well, while a second patient under similar circumstances develops an incurable myelitis. There is no reason for believing that the two cases are really anatomically diverse. There is much reason for clinically separating curable and incurable cases and for the conveniences of teaching, I depart from what might be considered scientifically strictly correct.

Let us pause a moment to see how sexual excesses produce spinal disorder. During the sexual act the nerve-centres, and probably especially the lower portions of the cord, are intensely excited. Many of the sexual movements are undoubtedly reflex, and the final orgasm is accompanied by a paroxysm comparable to a spinal epilepsy. It cannot but be that there is an active hyperæmia of the nerve-centres during the act, and a decided exhaustion of these centres follows as the result of their intense functional activity. Normally, the congestion subsides and the exhaustion is recovered from without any evil results. But if the act be repeated at short intervals, the repeated congestion exhausts the vessels, so that there is developed a condition of chronic congestion along with a constantly-increasing exhaustion. The spinal centres are, therefore, placed under the most favorable circumstances for the development of a low grade of nutritive changes ending in degeneration and constituting one of the processes of inflammation. If a case is seen very early, before any decided change has occurred, we speak of it as sexual exhaustion, and the chances of affording relief are good. If, however, the structural degeneration has progressed, then we say the man has chronic myelitis, and the prognosis is very gloomy. Of course there is every shade between the extremes, and it is often impossible to locate exactly a given case.

White softening of the cord is a rare disorder, which it is very difficult, if not impossible to separate from the torpid form of chronic myelitis. It is even denied that there is such a disorder as a slow, non-inflammatory breaking down of the spinal marrow, but I have certainly seen at least one case in which, during life, the symptoms were simply a progressive loss of motor and sensory power in the lower limbs, ending in absolute paralysis of every spinal function, and in which, after death, the lower third of the cord was resolved into a whitish fluid or semi-fluid mass of detritus. If these cases can be clinically separated during life from those of true myelitis, it must be by the complete absence, even in the beginning, of pain and of spasm and of heightened reflex activity and the steady deterioration of the power of motion, and of sensation, and of the other less distinct functions of the spinal marrow.

Of *chronic myelitis* there are two chief varieties : one ending in hardening of the cord, the other in the development of a greater or less amount of softened tissue. In the first of these, *sclerosis*, the whole cord or any portion of it may be affected. The process may be compared to that which occurs in cirrhosis of the liver, consisting essentially of an excessive nutritive activity or chronic inflammation of the connective tissue of the cord, giving rise to a great increase of this tissue, and finally a destruction of nerve-cells and tubules by pressure. This series of changes is essentially very slow, and, for a long time, is associated with a condition of hyperæmia and functional excitement of the spinal centres ; hence, violent pains and spasms, separate or conjoined, and local or general, according to the seat of the lesion, often precede, and finally co-exist, perhaps for years, with the paralysis. It is also evident that there must be, during the earlier portions of the disease, an exalted state of reflex activity, and in some cases this exaltation lasts long after the patient has lost his ability even to stand ; moreover, in the majority of cases of spinal sclerosis the membranes share the disorder, and from the resultant irritation of the nerves, pain and spasm result.

In that form of chronic myelitis which results in softening and breaking down of the nerve-tissues, the membranes are rarely affected, and the cord itself is not, during any long period, in the condition of chronic hyperæmia and irritation seen in spinal sclerosis. Very early in the disease the nerve-cells commence a deterioration which lessens their functional activity. For the reasons just given, the onset of this form of chronic myelitis is a comparatively torpid one. The spasms, the pains, the heightened reflex movements of sclerosis, are almost wanting, or are present only for a short time, and in a mild degree. Further, while sclerosis is very often strictly confined to the posterior columns or to some other regions of the cord, the myelitis with softening usually affects the whole cord. Bearing these facts in mind, you will rarely have much difficulty in coming to a decision. Sometimes, however, the two varieties of myelitis co-exist, and the symptomatology is of a mixed character. Some of these cases are very puzzling, but when the symptoms are so mixed, it is safe to conclude that the lesion is also composite.

Cases of *sclerosis* may be divided into four classes: those in which the anterior or the antero-lateral columns are alone affected; those in which the posterior columns are diseased, and those in which the lesions affect both of those structures. In either case the symptoms arise from the disturbance of the functions of the affected part.

In the affection known as *loco-motor ataxia*, the seat of the lesion is in the *posterior columns*. Since the transverse fibres, the prolongation of the posterior nerve-roots are irritated very early in the disease by the contiguous inflammation, pain is one of the earliest symptoms. The pains are prone to appear chiefly in the neighborhood of the joints. Sometimes they are limited to a very small region, and are described as boring; sometimes they shoot with intense rapidity through the limb, and are compared to a lightning-like thrust with a white hot wire. In either of these varieties the agony is but momentary, but the intervals between the pains may be very brief, though, usually, they are at first long, and the patient may be free from suffering for hours, or even for days. The maximum intensity of the pains is generally at night. In the intermissions, the patient is entirely free from suffering, except in some cases, when he is plagued with constrictive trunkal pains.

The severity of the suffering, of course, varies very greatly, and some authors even assert that not rarely these pains are altogether absent. As the posterior columns are so arranged in bands or sub-columns that it is possible for a sclerosis to avoid the posterior nerve-roots, such cases may occur, but they are very rare; and I have no doubt, in most of the supposed instances, the patient has suffered from degeneration of the central gray matter, and not of the posterior columns. Sometimes to the pains in the limbs are added visceral neuralgias. Violent rectal, vesical, or urethral pains may accompany those in the limbs or may occur during the intervals, or may even for a time replace the more usual symptoms. Sometimes they are so violent and are accompanied by such evidences of functional excitement, as intense desire to urinate or defecate, violent rectal or vesical tenesmus, and even satyriasis, as to constitute a veritable crisis. Charcot has recently called attention to a very violent form of gastralgia, as sometimes present in loco-motor ataxia. Very suddenly the patient is seized with a pain, commencing in each side of the abdomen, but soon centring in the epigastrium. Simultaneously a pain radiates through the trunk from a centre situated between the shoulders; the pulse is usually much accelerated, rarely lessened in frequency; the vomiting is extremely violent and persistent; not rarely a violent cardialgia develops itself, and if, as is usually the case, the limbs are traversed by atrocious darts of pain, the acme of human suffering is reached. The attack may last for several days, and is prone to be repeated at somewhat regular intervals.

Whenever, gentlemen, pains of the character which I have just portrayed occur repeatedly and persistently, you should suspect very strongly the existence of loco-motor ataxia. And remember, an unconquerable lesion may progress so slowly as to take years for the development of other symptoms than pain.

Nothing is more common, than for these pains of loco-motor ataxia to be mistaken, at least for a time, for rheumatism or neuralgia. Only a few weeks since, a clergyman of some prominence was brought to me by his family physician, with a statement that he and others had been treating the gentleman for some months for sciatica, but without affording him any relief. I at once made the diagnosis of spinal degeneration, and the subsequent progress has confirmed this. The points which decided my opinion will serve to illustrate well the general subject. They were, the affection of both legs, the absence of the focal pain-points seen in sciatica, the complete absence of tenderness over the whole course of the nerve—both upon direct pressure and upon motion—the presence of decided anæsthesia, and the long intervals which at first occurred between the pains.

When, in the period of pain, certain other symptoms develop themselves, the diagnosis becomes more easy. Prominent among these aids are certain indications of disease at the base of the brain, which may put you off, although they ought to put you on, your guard. These are indications of irritation, or, more usually, of paralysis of the cranial nerves. Of these nerves, the oculo-motor and the sixth pair are said to be the most frequently affected; but the hypoglossal, the facial, the trigeminal, the auditory, and even the vagus may be implicated, and complete destruction of the optic nerve is sufficiently common. Dilatation or contraction of the pupil, diplopia, loss of hearing, paralysis of sensation or motion in the face, amaurosis, may exist separately or conjointly, and may lead you, if careless, to diagnose cerebral tumor. Often the ophthalmoscope will reveal a peculiar degeneration, whose characters I will not here dwell upon, of the papilla, instead of the choked disk of cerebral tumor; but sometimes this is wanting, and the diagnosis has to be made by the presence of the pains and the absence of distinct cerebral disturbance, such as failure of memory, headache, etc., aided by the slow, regular development of the symptoms, and by certain peculiarities of the amaurosis. Often the field of vision is concentrically or unilaterally sharply limited, and not rarely there is an early dischromaptopsis or loss of the power of distinguishing colors. This progressive color-blindness usually first affects in order green, red, violet; the yellow and blue being longest distinguished.

Sooner or later in loco-motor ataxia the exudation presses upon nerve-fibres, and then, to the signs of irritation, is added failure of function, which is evinced in an anæsthesia that affects not only ordinary sensibility, but also all the particular forms of sensation. If the disease extend sufficiently upwards, the patient is no longer able to judge of weights by lifting them ; and if the hand or foot be laid upon a flat surface and weights be placed upon it, the sufferer, unaided by vision, is unable to determine whether the weight be two or ten pounds. It is in this disorder especially that is seen the peculiar retardation of the passage of sensation already spoken of. In these cases, when the patient walks, he sees his feet touch the floor, but does not, until after a considerable interval, also feel them on it. The commissural fibres usually are also pressed upon by the exudation, and coördination is soon affected, and, after a time, becomes impossible, so that the patient is unable to walk. You judge that the paraplegia is apparent rather than real, from the fact that when lying upon the bed the patient can kick with the greatest force. It is a very common test in suspected loco-motor ataxia to try whether the patient can walk in the dark. This fact, gentlemen, that a man cannot walk with his eyes shut, is no proof that there is loss of coördination, or that he has locomotor ataxia. It simply shows that general sensibility is abolished, and this may occur from disease of the central gray matter of the cord. A man walks in the crowded street intently reading a book, or hurries along in absolute darkness, or in his sleep, with closed eyes, mounts to the roof, because the requisite extremely-delicate combinations of muscular action are in the absence of vision presided over, or, more correctly, guided by feeling. The loss of the ability to do these things depends upon the destruction, not of coördination, but of that sense of touch, which, in the darkness, is the sole guide of muscular movements. I have seen this inability to walk in the dark, when the central gray matter of the cord, so far as could be judged during life, was alone affected.

When. the *anterior columns* are affected with sclerosis, the symptoms produced are spasms, and general or local mild muscular contractions, with heightened reflex activity, without pain and anæsthesia, but with a steadily progressive loss of power, which finally conquers, perhaps after the lapse of years, the more active symptoms.

The case which I now show you, I believe to be suffering from a spinal sclerosis. The young man, aged twenty, now bed-ridden, tells us that up to the age of eleven he was a very active boy; but that in the fall of 1865, after recovering from an attack of some acute disorder, he noticed that in walking, he trod upon his toes. This tendency increased until he was unable to walk at all, although he could run or trot well enough. The moment his feet touched the ground, his heel flew up, and his body was thrown forward as in running. In my colleague Dr. Pepper's ward, in the Philadelphia Hospital, there is a man now just in this condition. The peculiar movements evidently depend upon a condition of excessive activity of the spinal cord. The moment the foot is planted for the purpose of executing the largely reflex movements of walking, all the leg muscles are thrown, not into normal, but into irregular spastic contraction, and the calf muscles being very much stronger than their antagonists upon the front of the foot, of necessity, up goes the heel.

But to return to the history of our patient; during 1866, his difficulty of walking increased, and he had occasional nocturnal incontinence, but not until the beginning of 1872 was he conscious of very serious loss of muscular power, and by the end of the year he was helpless. Now he lies with his heels drawn up, feet inverted forcibly, the knees rigid and in forcible contact. He can flex the thighs on the body, but cannot raise the heel off the bed, and can scarcely bend the toes. His feet are inverted, his knees closely jammed together owing to the violent, tonic contraction of the adductors. In the arms a considerable amount of power is retained, but the fingers are habitually in some position of rigid extension. An electrical examination shows that no muscle has lost its electro-contractility, and in those affected with spasm, this contractility is apparently even greater than normal. This case at first sight resembles one of meningitis, but there is a distinct history of absence of pain ; a history which we must presume to be correct, although we cannot be certain of it. The symptoms which I have detailed, certainly point to a sclerosis of the anterior columns, but notice that, as I put the points of an æsthesiometer upon his limbs, he cannot distinguish them as two points until they are separated at least nine inches. This is true both of his arms and legs. No disease of the anterior column; not even their total destruction, could produce this amount of loss of sensibility. The absence of pain shows that the posterior columns have escaped a general implication, and, if his history be relied on, we are forced to the conclusion, that the gray matter of the cord is implicated, or that portions of the posterior columns are involved.

There are certain cases of spinal disease, in which the chief symptoms are an intense state of excitement of the reflex activity, resulting in spasm so violent and persistent that the patient is unable to walk, although there is sufficient retention of muscular power.

In such a category may be placed the affections described by Brown-Séquard under the term of *spinal epilepsy*, and E. C. Seguin under the name of *tetanoid pseudo-paraplegia*.

In spinal epilepsy, "either spontaneously or after an external irritation, the lower limbs are often moved violently, or become perfectly stiff; sometimes they are drawn up forcibly in a state of flexion, the back part of the foot pressing against the hip joint ; sometimes the thighs are drawn violently one against the other by a spasm of the adductor muscles, and press very hard against the testicles ; in other cases, the flexor and extensor muscles contract alternately with great violence, and, after a few minutes of great shaking a rigid condition appears, which, after a time, is followed by relaxation and quietness." In tetanoid pseudo-paraplegia the attempt to walk produces tonic spasms of the leg-muscles, which renders walking exceedingly difficult. On rising from a bed or chair the patient oscillates a good deal, and seeks to re-establish his equilibrium by separating his feet, and bending his body forward. The knees remain extended, the feet are more or less inverted, and in walking are simply pushed along the floor.

I do not think either of the states just spoken of is worthy of a distinct place in a Nosological Catalogue, they are simply the results of an exceedingly exalted reflex activity. In many of the cases the exact pathology of the condition has not been thoroughly made out. Brown-Séquard believes that spinal epilepsy is due to a *local myelitis*, and in my arrangement, for the sake of convenience I have used this name to cover both of the states under consideration, although I am by no means sure of its scientific correctness. In a large proportion of the cases the original lesion has been shown to be a Potts' disease or a spinal tumor, and it seems plausible that the excessive reflex excitability has depended partly upon a cutting off of the inhibitory influence of the brain, and partly upon an irritation of the central gray matter propagated from the point of pressure.

We come, finally, gentlemen, to the consideration of these cases of organic spinal disease, in which paraplegia coexists with marked tremors or tremblings.

Paralysis agitans is probably not a disease of the spinal cord. Its pathology is not thoroughly determined, but the affection has in some cases appeared to depend upon a lesion of the pons varollii. The disease does certainly, however, give origin to an apparent if not a real paraplegia, and we must, therefore, consider it here. This disorder may occur suddenly, as the result of a profound moral agitation, or it may develop very slowly. In either case it is characterized by the existence of continuous tremors accompanied by muscular enfeeblement. The diagnosis is so evident, that it is not necessary here to draw even a sketch of the disease. In *multiple spinal sclerosis*, also, general tremors with muscular enfeeblement, without other symptoms, are the pathognomonic features of the disorder. The difference between the two diseases lies in the fact that in multiple spinal sclerosis, the *tremors* occur only during voluntary movement. When the patient is quiet the muscles remain perfectly at rest.

Some of you may remember a man, whom I lectured on last spring at this clinic, as a typical example of multiple spinal sclerosis. He sat in a chair perfectly quiet, but the moment he seized a pen, his right hand, and in a short time also his left hand, began to shake violently. When he attempted to rise, his feet shook to and fro beneath him; tremors seized upon his arms as he attempted to walk, and the general trembling became so excessive, that, without aid, progression was impossible. In that case cerebration was normal, but often the lesion compromits the brain, and to the spinal manifestations are added cerebral symptoms.

In concluding, gentlemen, this birds'-eye view of spinal diseases, I may be allowed to express the desire that to each of you it may prove a useful skeleton, to be by future study and experience filled out and rounded into a perfect form.

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ON THE NATURE OF THE GOUTY VICE;
ITS MANIFESTATIONS AND TREATMENT.

BY

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THE logical sequence to the didactic description I have given you of the various primary lesions of the skin, would be a consideration of the causes, local and constitutional, which determine these lesions. It would be impossible for me, however, in the brief time allotted for your instruction in this class of diseases, to give you anything like a complete review of all the causes which may determine lesions of the skin, and I shall have to content myself with alluding to most of them only in connection with such cases as may be presented at the Clinique.

There are some general considerations, however, respecting the general pathology of skin diseases, which I wish to bring before you early in the course, because I shall have frequent occasion to make use of them in the diagnosis and treatment of the cases which are likely to constitute the majority of those which will present themselves to us. I shall ask your attention, therefore, in this and several succeeding lectures, to a general consideration of the constitutional vices, or diatheses, as they are sometimes called, which present in their evolution a great variety of morbid phenomena, and among them, numerous and important affections of the skin. These constitutional vices are *Gout*, *Syphilis*, and *Scrofula.*

In thus presenting to you the diathetic origin of many of those lesions of the skin which do not recognize a local cause, I ought to premise, that though there is no question among dermatologists as to the specific origin of the tegumentary lesions which occur in the evolution of syphilis, there is a radical division of opinion, as to the influence which gout and scrofula have, in the production of skin diseases. The French School, of which Bazin and Hardy may be regarded as the modern representatives, espouses the Humoral Pathology of many cutaneous lesions, while the German School, of which Hebra is the renowned exponent, may be said to represent in dermatology the pathology of the Solidists, who locate the origin of all lesions in the tissues themselves.

The objection brought against the recognition of diatheses or constitutional vices, that their nature cannot be definitely demonstrated by the isolation of a virus or materies morbi, and that there is no common consensus as to what constitutes the special manifestations of these several vices, is not without force. It cannot be denied, however, that clinical observers in all times, particularly those who are not strictly specialists, have correlated many morbid conditions in such a way as to justify the hypothesis of definite constitutional states. This correlation has been based upon the co-existence in individuals and families of various morbid manifestations which are allied by common characteristics, often by hereditary transmission and especially by the results of definite treatment. The utility, for example, of mercury and iodine in syphilis, of iron and cod liver oil in scrofula, and of the alkalies in gout, all point to the existence, in these vices, of some morbid entity in each, which lies at the root of their various local manifestations. As most of you will have to study skin diseases from the standpoint of general practitioners, and not as specialists, your experience will, I believe, confirm the existence of certain relations between lesions of the skin and those of other organs, which make the recognition of the constitutional vices I have mentioned a reasonable and practically valuable hypothesis.

I propose to-day, therefore, to ask your attention to gout as the determining cause of a class of morbid phenomena, among which various affections of the skin hold a prominent place—and first, I need hardly tell you that I do not use the term "gout" in its restricted sense, as applied to a special variety of articular affections, but in the more comprehensive signification with which the term is used to express a constitutional vice.

The evidence upon which the recognition of this diathesis rests, is derived partly from the revelations of organic chemistry, and partly from the clinical history and treatment of gouty diseases. The evidence furnished by physiological chemistry relates to the changes through which the food passes in the process of digestion and assimilation, and I must ask your indulgence while I review, as briefly as possible, the physiological transformations of our food; for it is in the interruption of these transformations, that we trace the *fons et origo* of the gouty habit. Our food, as you know, is composed of albuminous and carbonaceous substances, and its destiny is the renewal of our tissues and the conversion of the potential force, which it represents, into active energy. Every atom of albuminous or nitrogenous food that we consume passes, in the process of digestion and assimilation, through a series of complex changes, until, having expended itself in the renewal of tissue or the development of force, in the form of heat, or mechanical or some other form of vital energy, its ash, or useless residuum, leaves the body, through the emunctories, in the form of nitrogenous excreta. In the same way, every atom of carbonaceous food passes through a regular but less complex series of transformations, until, having served its purposes, it is reduced to carbonic acid and water, which pass off through the lungs and skin. Now these transformations are accomplished through the medium of the oxygen in the air we breathe :—the process is one of combustion, and our bodies are, in one sense, neither more nor less than furnaces, which have within themselves the capacity for keeping themselves in a state of integrity and continual repair, and at the same time of converting, through the process of oxidation, the latent energies stored up in the food-fuel into the various manifestations of vital force.

In the highest conceivable state of health, therefore, the combustion of our food is complete, and the useless waste excreted passes again to the earth, and into the atmosphere, to enter upon that series of synthetical changes by which the ammonia, the carbon, and the salts are prepared for the development of vegetable life. Unhappily, however, there are often many checks and interruptions in the harmonious evolution by which the phenomena of animal and vegetable life are sustained. The most remarkable illustration of the check which the process of assimilation may suffer in animal bodies, and thus cause a serious departure from the state of health, is in the disease known as glycosuria, upon which I have recently had the pleasure of lecturing to you.

In this malady, as you remember, the presence of sugar in the urine was found to be due, sometimes to an arrest of the transformation of the amylaceous and saccharine elements of the food at the formation of glucose, and sometimes, in the more serious form of the disease, to the reconversion of the glycogen, formed in the liver from albuminous as well as carbonaceous elements, into sugar, its normal disintegration being checked. In gout, which in its etiology presents a striking analogy to diabetes, a similar check occurs in the conversion of the nitrogenous elements of the food. Instead of being converted into the soluble and useless excrement known as urea, the oxidation is arrested at the formation of the penultimate uric, or lithic acid, a very sparingly soluble substance, which, combining with the alkaline bases, principally the soda, circulates in the blood and is not only eliminated in excess by the kidneys, but diffuses itself through the tissues, giving rise, as we shall see, to many serious and painful forms of mechanical irritation.

I need not now recall to you the important office of the liver in the conversion of the amylaceous and saccharine foods, but I wish to call your attention, briefly, to the important function of this organ in the disintegration of the albuminous foods. Formerly, it was supposed that the only office of the liver was to secrete bile ; the discovery of its glycogenic function by Bernard added largely to its physiological reputation, and now the tendency of modern investigation is to make it responsible for the formation of urea. This hypothesis has been very fully developed in the past year by Murchison in his Croonian lectures. The principal arguments in favor of this idea are : first, that one of the most constant signs of functional derangement of the liver is an imperfect formation of urea, as indicated by the deposit of lithic acid, lithates, and an excess of coloring matter in the urine ; second, when a considerable portion of the liver is destroyed by disease, the urea discharged is greatly lessened or may entirely disappear ; and third, that in acute atrophy of the liver, in which the secreting cells undergo rapid and complete degeneration, all trace of urea may disappear from the urine, its place being taken by less oxidized substances, such as leucin and tyrosin, which are also found in large quantity in the hepatic tissue, as if they marked the arrest or modification of the transformation of albumen.

The gouty diathesis, therefore, in its chemical aspect, consists essentially in an interruption of the process of oxidation of the albuminous elements of the food, the check taking place at the formation of lithic acid. This acid combines, as we have said, with the alkaline bases in the serum, forming lithates which circulate in the blood and diffuse themselves through the tissues. This excess of lithic acid has been very properly termed lithæmia. In its pathological relations, the gouty diathesis would appear to originate in a derangement of that function of the liver, by which it disintegrates the albuminous foods and converts them into urea.

It seems probable, from the frequency with which diabetes occurs in gouty subjects, and from other evidences, to which we shall hereafter allude, of the feeble capacity which the latter exhibit for the digestion of carbonaceous foods, that the defective oxidation which seems to be the essential element in the malassimilation of sufferers from the gouty diathesis, is not confined to the albuminous foods, but is manifested also in the imperfect combustion of the non-nitrogenous aliments.

In giving you this sketch of the nature of the gouty diathesis, I ought to tell you that there are objections to it which still stand in the way of its universal acceptance, but the hypothesis seems to be justified by physiological facts and serves better than any other to link into harmonious union the phenomena of gout, and to form a rational basis for their successful treatment.

Let me now ask your attention to a brief consideration of the etiology of this diathesis. It is an hereditary and an acquired vice. As in form and feature, and in moral and intellectual traits we are patterned after our progenitors, so, in the performance of our vegetative functions, we perpetuate their peculiarities. As an hereditary vice, gout manifests, like other hereditary diseases, the phenomenon of atavism and the limitations of sex and age, though in these latter respects it presents considerable irregularities. It is commonly a disease of adult life, though we may often recognize in early childhood and during adolescence, the evidences of feeble digestive power, and the tendency to lithæmia out of which its more characteristic phenomena are developed. A careful study of the digestive weaknesses of young children will frequently illustrate the inheritance of the parental liver, just as the observation of their physical and mental traits reveals a likeness to their prototypes. It is not only, however, in a feeble digestion that children manifest the inheritance of the gouty vice; they may be the subjects of genuine articular gout, and repeated observation has led me to believe that the children of gouty parents are especially liable to acute inflammatory rheumatism. The tegumentary structures, the skin and mucous membranes, manifest, perhaps more frequently than any other tissues in young subjects, the effect of inherited gout. The liability of these structures to all forms of parasitic invasion, their feeble resistance to mechanical, medicinal, and chemical irritants, and especially their susceptibility to the poisons which produce exanthematous lesions, are familiar facts, and afford a sufficient reason why young subjects should be especially liable to those affections of the skin and mucous membranes which depend upon the circulation in the blood and diffusion into the tissues, of those peccant matters which result from an imperfect digestion of the food.

As an acquired vice, gout may be said to depend upon the conditions which bring about an excess of lithic acid in the blood and tissues. These conditions are, first, the ingestion of an excessive quantity of animal and vegetable albuminous foods—more than can be consumed by the ordinary supply of oxygen ; and, second, a diminished supply of oxygen, the nitrogenous foods not being in excess of the requirements of the system. If the amount of food daily ingested is more than is needed for the repair of the tissues and the evolution of the required heat and the other forces to be provided for, the excess is pretty certain to escape complete combustion. There is a loss of balance between the supply of oxygen and the matters to be oxidized, hence the accumulation in the blood and the diffusion into the textures, or the excessive discharge through the emunctories, of imperfectly oxidized waste. This is the common origin of the gouty diathesis among the luxurious classes who live to gratify their appetites, rather than to develop, by useful toil, the energies stored up in their food. But gout, likewise, comes to those whose confinement in close and ill-ventilated dwellings and workshops deprives them of an adequate supply of oxygen for the complete conversion of the food they consume, and the same manifestations of mal-nutrition may thus occur in the over-fed drones of society, and in the industrious but poorly housed laborers.

Indeed, the circumstances to which the chemistry of nutrition justifies us in ascribing the gouty diathesis, are such as affect persons in all classes of society, and arise as well from the temptations which surround us, as from the ignorance or defiance of simple hygienic laws. Thus it happens that this constitutional vice, in its protean forms, is very much more common than is generally imagined, and is responsible for a large proportion of the organic and functional derangements of health which come under the observation of the physician.

It is hardly necessary to observe that in giving this chemical explanation of the origin of lithæmia, as the basis of the gouty vice, I do not intend to give you the idea that nutrition is so simple a process as this explanation might imply, or that the etiology of gout is all comprised in the theory of sub-oxidation. We may safely assume that sub-oxidation is the condition upon which lithæmia and glycosuria depends, but while sub-oxidation doubtless often results from a loss of balance between the food-fuel and the inspired oxygen, we should not forget that in so complex a machine as the animal organism, many circumstances may intervene to disturb the chemistry of nutrition. It would be impossible, within the limits of this lecture, to dwell upon the many predisposing causes of a faulty nutrition. The effects of organic lesions of the lungs, the liver, and the kidneys, of certain metallic poisons, as, for example, lead, and of nervous shock and exhaustion from various causes, are all circumstances to which, at some future time, I may allude, as predisposing causes of the imperfect evolution of the process of digestion, of which sub-oxidation is the direct and inevitable result.

Let me now ask your attention to a brief description of some of the more common functional and organic derangements of health to which gouty persons are subject. The earliest and perhaps the most frequent functional disturbance in the evolution of the gouty habit, is a flatulent and acid dyspepsia. This appears to depend, in the majority of cases, upon an acid fermentation of the carbonaceous elements of the food, and notably of the sugars and starches. This conclusion is based upon the prompt and marked relief of the symptoms of this dyspepsia which follows upon the diminution or withdrawal of these elements from the diet.

The direct and reflected effects of this kind of indigestion are numerous and very varied. The direct effects are a sense of weight and fullness at the epigastrium after eating, with acid eructations, and frequently intestinal borborygmus, accompanied with colic, and alternating constipation and diarrhœa. The reflected irritations are manifested in the palpitation and irregular action of the heart; in sighing respiration, and oppressed breathing; in a sense of heaviness and muscular weariness, and not infrequently, in persons of neurotic temperament, in the multiform nervous symptoms which characterize hypochondriasis and hysteria.

I should mention, as a circumstance of great importance in the history of this gouty form of dyspepsia, that the subjects of it are especially intolerant of all fermented preparations of alcohol. They are almost invariably made uncomfortable by moderate use of beers or wines of any description, and persistent indulgence in them is very certain, sooner or later, to culminate in the condition commonly described as biliousness, which is the vulgar term for the combination of symptoms I have just detailed to you.

Another and not uncommon disturbance to which the gouty dyspeptic is subject, is a frequent and painful micturition, sometimes accompanied by a heavy dragging pain in the course of the ureters. This vesical irritability and neuralgia in the urinary passages is associated with certain changes in the physical and chemical qualities of the urine. I can only briefly allude to these changes. They affect principally the reaction and the proportion of the solid constituents. The reaction of the urine is intensified in the lithæmic state by the excess, in the urine, of the acid salts, the phosphates and urates, as well, probably, as of the free acids, the lactic, oxalic, and acetic, upon which its normal acidity depends. This acidity is aggravated by a highly animalized diet, and is most troublesome after the completion of the primary digestion. The effect of a meal being always, as Bence Jones first pointed out, to depress the acidity of the urine, while its remote consequence is to increase it. Hence frequent micturition from excessive acidity is most apt to be marked during the later hours of a long fast, and will be found generally to bear the relation to the meals that has been suggested.

Frequent and painful micturition, however, when not dependent on organic disease of the pelvic organs, is commonly associated with the presence in the urine of deposits of crystalline sediments of urates, oxalates, and uric acid. These deposits sometimes take place in the urinary passages, giving rise to gravel, and to renal and vesical calculi, and sometimes at varying intervals after the passage of the urine from the bladder. The relation of excessive acidity to the formation of these deposits is exceedingly interesting and important, but need not be dwelt upon here. Temporary derangements of the urine, such as I have described, are or course sufficiently common in acute dyspeptic attacks, and in febrile conditions, but where they are persistent, and especially where they are associated with symptoms of crystalline deposits in the tubuli uriniferi and the pelvis of the kidney, and in the bladder, they betoken the condition known as lithiasis, and become one of the most significant and important indications of the gouty vice.

The subjects of the gouty diathesis are also liable to certain functional derangements of the nervous system, which deserve special mention. Many of the nervous phenomena of gout are doubtless associated with an organic lesion of the sheaths of the nerves, constituting what is called neuritis, but there is a class of neuralgic symptoms which differ from those of true neural affections in the absence of tender points, and in not following the course and distribution of nerves, which are reasonably supposed to depend upon the central and peripheral irritations caused by lithæmia. Some of these have already been mentioned in the description of the subjective sensations of the gouty dyspeptic; others are apparently myalgic, and are commonly described as muscular rheumatism. Frontal headache, pain in the back of the head and neck, pain in the eyeballs, with the other indications of the so-called æsthenopia, burning sensations in the palms and soles, numbness of the hands and forearms, pain in the tendo achillis and the dorsum of the foot, are some of the more common dysæsthesiæ complained of by gouty subjects.

We may now briefly consider some of the organic affections provoked by the gouty vice. The tendency to local congestions in the gouty dyscrasia, is a pathological fact of primary importance. Whether this tendency arises from the irritation of the vasomotor or trophic nerves by a poisoned blood, or is due to a disturbance of that force in the capillary circulation which is dependent upon the chemical affinity between the blood and the tissues, is still a matter of speculation, but the clinical fact is unquestioned, that sufferers from gout are especially liable to local congestions. These congestions are particularly prone to occur in the fibrous structures, in the synovial membranes, in the tendons and aponeuroses, in the nerve-sheaths and cerebro-spinal envelopes, in the cornea, the sclerotic, and in the fibrous stroma of the parenchymatous organs. In the joints they are frequently excited by mechanical injury. Hence, they are most common in the small joints of the hands and feet, which are exposed to more frequent strain and injury than the larger joints, and which are doubtless also rendered more susceptible by their distance from the centre of circulation. When the joints are rendered more vulnerable by inheritance or by repeated attacks of gout, other causes may provoke a seizure, such as a debauch, a fright, or the nervous exhaustion incident to prolonged anxiety or fatigue. The pain which is incident to arthritic gout, is doubtless principally due to the pressure made by the distended vessels upon the sensitive nerves, and the difficulty with which swelling occurs in these structures, and the sudden and almost complete subsidence of pain when swelling has occurred, is the result of the pressure having been taken off from the distended vessels by the free transudation of serum into the surrounding connective-tissue. The consequences of these articular inflammations are seen in the thickening, due to the hyperplasia of the connective tissue around the joints, and to the deposit of the characteristic chalky concretions of urate of soda.

The tegumentary structures, the mucous membranes, as well as the skin, are also the seat of morbid changes in those who inherit or acquire the gouty vice. The frequent association of chronic bronchitis with articular gout has been noticed by numerous observers, and has been ably presented in its clinical aspects by Dr. Greenhow of London. I have had frequent occasion to verify this connection of catarrhal affections of the air-passages with the other manifestations of the gouty diathesis, and where this form of bronchitis occurs in persons of neurotic temperament, the complication of spasmodic asthma is, I believe, not infrequent. A gouty or rheumatic form of conjunctivitis and iritis is generally recognized.

The gouty lesions of the gastro-intestinal mucous membrane are very common, and constitute the determining cause of many of the more chronic forms of dyspepsia. They consist in localized hyperæmia, and catarrhal inflammations of different portions of the mucous lining of the alimentary canal, and are probably caused primarily by the irritation of disordered secretions, or by the disturbance of the capillary circulation, induced by a blood overcharged with the elements of imperfectly disintegrated food.

In the mouth, these lesions are recognized in the swollen, pasty, and indented tongue with enlarged papillæ, in a tendency to aphthous ulcerations after slight abrasions, and in pharyngeal catarrh. In the stomach they are present in the form of a sub-acute gastritis, as indicated by epigastric tenderness and pain, by pyrosis and nausea, and flatulence with its attendant reflex disturbances.

In the intestinal tract they constitute the cause of a variety of morbid phenomena, giving rise, for example, to jaundice, in duodenal catarrh, to colic, flatulent distension, and constipation, or constipation alternating with diarrhœa. Hemorrhoidal engorgement, which so frequently accompanies the signs of intestinal disturbance just mentioned, is one of the lesions to which gouty subjects are especially liable.

I would also allude here to the uterine derangements, both functional and organic, which have occasionally seemed to me to be aggravated by, if they are not sometimes dependent upon, the existence of this vice. Irregularities in menstruation, especially painful and profuse menstruation, are certainly not unfrequently associated with the dyspeptic symptoms and other signs of disordered nutrition, which we have described as characteristic of gout. I am also inclined to believe that uterine and vaginal catarrhs are very frequently of gouty origin, and that topical medication in such cases is rendered much more efficient by constitutional treatment directed to this vice.

In this connection I might also refer to the influence which the gouty vice exerts upon urethral catarrh. The tendency which gonorrhœa manifests in gouty subjects to pass into gleet, and the frequent repetition of the acute symptoms induced by imprudent indulgence in eating and drinking, are common observations in the experience of every physician. Not less interesting and important, as corroborative evidence of the gouty nature of certain forms of urethritis, is the occurrence of the so-called gonorrhœal rheumatism. That this is a gouty affection I have several times been able to determine most satisfactorily by the existence of a distinct gouty history, and once by the detection of uric acid, in notable quantity, in the blood.

The lesions of the skin which occur in gouty subjects may be divided into those which depend upon sudden and transient disturbances of the cutaneous circulation, and those which are the consequence of prolonged hyperæmia.

Examples of the former class are observed in some of the forms of erythematous lesions, as, for instance, in urticaria and in that more rare and curious affection of the same fugitive character, which occurs generally about the face, in which sudden swelling takes place in the eyelids, closing the eyes, or in the cheek or lips, or perhaps in the tongue or soft palate, causing marked but transient deformity, and oftentimes considerable inconvenience and suffering. In these forms of erythema the hyperæmia is temporary, lasting only a few hours and leaving no trace. It should also be remarked that these lesions in gouty subjects are generally excited by indigestion and especially by the irritation, either direct or reflex, produced by certain articles of diet, such as certain kinds of fish, or fruits, or wines. The same idiosyncrasies are observed in such subjects in regard to the susceptibility to external irritants. Allied to the erythematous affections just mentioned are the more persistent lesions known as erythema nodosum, and the purpura rheumatica, which are commonly accompanied with articular symptoms, and are generally recognized as gouty or rheumatic in their origin. Boils and carbuncles should also be mentioned as affections of the skin, which are frequently observed in persons having the gouty diathesis. They are associated with the other derangements of nutrition which seem to be dependent upon the circulation in the blood of the effete products which result from imperfect oxidation, not only of the albuminous but of the carbonaceous elements of our food. The frequency of carbuncular inflammations in diabetes, for example, is a well-recognized fact.

The other division of gouty lesions of the skin, those in which a more or less persistent hyperæmia is present, is exceedingly frequent, and includes some of the most important and troublesome forms of cutaneous lesions we are called upon to treat. In the more acute varieties of these lesions, as for example, in erysipelas, in acute eczemas, and in the erythema which sometimes accompanies articular gout, the inflammatory process is active, is attended with more or less constitutional disturbance, and runs a definite course, like other phlegmasiæ. In the subacute or chronic forms, the hyperæmia results in a deranged and riotous sort of nutrition, causing hyperplasia of the different tissues entering into the structure of the skin and resulting in excessive production of epidermal cells, and in infiltration and thickening of the derma and subcutaneous connective tissue.

The cutaneous lesions of this nature most frequently associated with the gouty vice, are psoriasis, the chronic forms of eczema, and the different varieties of acne. It should constantly be borne in mind that in speaking of these affections as gouty, it is not to be inferred that they are always gouty in their origin. There are comparatively few lesions of the skin that are pathognomonic of specific constitutional vices. That gout, syphilis, and scrofula, however, do impress certain peculiarities upon the different primary lesions of the skin is undoubtedly true, but it is mainly by a consideration of the history of the patient, and the concomitant symptoms, that a differential diagnosis is to be made of the diathetic condition which determines the lesion.

The peculiarities impressed by the gouty habit upon psoriasis are: 1, A tendency to localization upon the scalp and the genital organs, and in persistent and often symmetrical patches upon the limbs. 2, A disposition to assume, in some instances, an eczematous character, as indicated by a deeper induration of the derma than ordinarily obtains in syphilitic and scrofulous psoriasis, and a certain amount of serous exudation ; and 3, A remarkable chronicity. Similar peculiarities are observed in gouty eczemas which localize themselves upon the hands and feet, upon the scalp and about the ears, in the flexures of the joints and upon the scrotum and perineum. I have several times observed, in gouty subjects, a single patch of eczema, generally upon the leg, below the knee, lasting for years, now and then taking on a more or less acute character, in which the derma has become greatly thickened and the subcutaneons connective tissue involved so as to give the affected skin a sclerematous appearance and consistence. The chronicity of eczemas in gouty persons is a marked characteristic, as is also their proclivity to temporary aggravation from derangements of the general health or undue mechanical irritation.

The varieties of acne, which are commonly associated with the gouty vice, are acne rosacea and acne indurata. In these lesions we observe the same tendencies to localization, to venous congestion and tissue hyperplasia, and to chronicity. They also are marked by the proclivity observed in other gouty lesions to temporary and repeated aggravation from reflected irritations, especially those connected with defective alimentation. I shall have occasion to call your attention repeatedly to the connection of these follicular affections with the gouty form of dyspepsia, and to the imperative necessity of recognizing this connection if you would treat these diseases successfully.

These, in brief, are the local peculiarities of the chronic lesions of the skin which are of gouty origin. You will observe that in their tendencies to localization, to hyperplasia of the epidermal and connective tissue, and in their chronicity, they present striking analogies to the chronic catarrhal affections of the mucous membranes which occur in gouty subjects, and also to the subacute articular affections which result in such subjects in thickening of the fibrous structures about the joints.

The subjective symptoms which accompany these lesions are especially characteristic. The burning and itching are sometimes the source of acute and most wearisome suffering. This is due partly to the acuteness of the disease and the sensitiveness of the locality affected, but, as in all diseases accompanied with pain, they vary probably according to the nervous constitution of the patient.

Among the gouty lesions of the parenchymatous organs, which are observed with unusual frequency in persons of gouty habit, are cynanche tonsillaris, or quinzy sore-throat, pneumonia, and the chronic form of nephritis which results in the granular or fibrocystic degeneration of the kidney, and the cirrhotic disease of the liver.

Cynanche tonsillaris is one of the diseases of adolescence and early adult life which, in my experience, is often associated with inherited gout and is sometimes accompanied by or alternates with articular symptoms. Acute pleuro-pneumonia, of the croupous variety, has also seemed to me to occur more often in persons whose history presents evidences of the gouty diathesis, and I am happy to observe that so distinguished an authority as Sir James Paget, in his recently published clinical lectures, alludes to the occurrence in his own person, of several attacks of pneumonia, which he believed to be rheumatic or gouty in their character.

Of the gouty nature of the cirrhotic kidney and liver there can be little question. Their frequent occurrence in persons who inherit gout, or whose habits have tended to induce it, and whose previous history presents unmistakable traces of gouty affections of other organs, furnishes insuperable evidence of their gouty origin. It is important and interesting to observe, that in these affections, as in those of a similar origin in other organs, the connective tissue is the special seat of lesion.

There are certain concomitant lesions associated with the gouty kidney which I should not omit to mention. I allude to the thickening of the muscular coat of the arteries and to the atheromatous affection of the arteries and valves of the heart, with the attendant ventricular hypertrophy. Equally worthy of mention are the accidents of embolism and arterial thrombosis which are the occasional sequences of these lesions.

I have thus briefly and, I fear, very imperfectly described the functional disorders and the principal organic lesions of the articular, tegumentary and parenchymatous structures observed in the evolution of the gouty vice. Before proceeding to the consideration of its treatment, I must ask your attention to the principles which should guide you in the diagnosis of this constitutional disease.

In the first place it is to be remembered that gout, in its articular form, and it is probable that the same is true of most of the manifestations of this vice, is a disease of adult life. This is certainly true of acquired gout, but experience has led me to believe that premonitions of inherited gout sometimes manifest themselves at a very early period of life, in the form of functional derangements of digestion and in a liability to some of the more transient and superficial affections of the skin. I have also been led to suspect, as I have before remarked, from the investigation of family histories, that acute inflammatory rheumatism is a disease to which the children of gouty parents are especially liable. As a rule, however, it may be stated that the evolution of inherited as well as of acquired gout does not begin until the commencement of adult life, when the process of nutrition, having accomplished the work of growth and development, becomes less active and is limited to the maintenance of tissue life, of animal heat, and the various forms of vital energy. The element of age, therefore, is, with the probable exceptions I have mentioned, an important one in the diagnosis of a gouty affection.

It should be observed also, that we find the gouty vice more frequently in men than in women—as an acquired vice, very much more frequently, for reasons which are creditable to the weaker sex; as an inherited vice it may not be more common in men than in women, though in the latter it probably often remains latent, partly from their more temperate habits and partly, perhaps, in consequence of the protective influence of menstruation. This idea was embodied in one of the aphorisms of Hippocrates, and the greater frequency of gouty affections in women at the menopause, is rather confirmatory of the truth of the observation.

The most important step, however, in the diagnosis of the gouty vice lies in a careful and painstaking review of the patient's health and habits and those of his ancestors. In seeking, for example, to substantiate the gouty nature of a cutaneous lesion, you must look for some of the concomitant functional and lesional derangements which are characteristic of gout. If the lesion be gouty, you will very certainly find, either in the individual himself, or in his direct or collateral ancestors, the evidence of these derangements. The proof derived from inheritance will relate to the existence in the direct or collateral ancestors of arthritic diseases, cutaneous and catarrhal affections, asthma and renal disorders. Frequently valuable information, corroborating a suspicion of this vice, may be obtained by seeking the causes of death in the family, for these are sometimes known, when little can be learned of the family diseases. Death from "heart disease," "Bright's disease," "dropsy," or sudden death from apoplexy, are always significant circumstances in the history of the gouty diathesis, just as death from phthisis is presumptive evidence of a scrofulous taint. I need not dwell upon the importance of the proofs derived from heredity in their relation to the formation of a correct judgment, in regard to prognosis and treatment, as well as diagnosis.

In the history of the individual, you will have first to consider his habits of life, in their etiological relations to the gouty diathesis.

From what has already been said you will recognize the importance of a careful investigation of the causes of acquired gout. Excessive indulgence at the table, combined with indolence, and sedentary or indoor occupations, and especially with the immoderate use of fermented drinks, are habits which must, sooner or later, induce the gouty vice in the most vigorous constitution. Having investigated the habits of your patient, you will direct your attention to the ordinary derangements of his health. These will commonly manifest themselves in some of the functional disturbances which I have described, as indicative of the gouty taint, and which generally precede the development of tissue changes. The most frequent derangement is a tendency to what is usually described as biliousness, or a malaise which is accompanied with physical languor and mental depression, with frontal headache and muscular pains, with pyrosis and flatulence, with constipation, or constipation alternating with diarrhœa, and especially with the presence in the urine of lithic and oxaluric deposits.

In addition to these functional derangements, you may find some lesional evidences of gout, and these should, at least, always be carefully sought. Subacute or acute articular attacks, with consequent deformity of the joints or contracted tendons, chronic bronchial catarrh, asthma, transient or persistent cutaneous lesions, renal colic and vesical catarrh are all organic affections which frequently recognize a gouty origin, and the occurrence of one or more of these transformations of the vice in the individual or his family should attract attention, and may reasonably confirm the suspicion of the gouty habit.

TREATMENT.—The treatment of gout, as a constitutional vice, is based upon principles deduced from the chemico-physiological theory of the process of nutrition, which I have described to you. If the transformations in the disintegration of food be the result of oxidation, it is clear that the derangements of this process are to be remedied by attention to the circumstances upon which thorough combustion must depend. These circumstances relate first, to the quantity and quality of the food; second, to the supply of oxygen; and third, to the use of remedies which are known to favor the process of oxidation and elimination.

I shall first ask your attention to the important relations of food, as regards its quantity and quality, to the treatment of the gouty vice. This will require a brief preliminary consideration of the quantity of food requisite for healthy nutrition. I need not tell you that it is impossible to arrive at anything more than an approximate estimate of the amount of food required for the nutrition of the body. The amount in the well-fed classes is probably always in excess of what is actually needed, though it is often not in excess of what is readily and quite perfectly digested. It must vary, of course, with the weight, with the habits and occupation of the individual, and with the necessity of protection against cold and heat.

The attempts to ascertain the proper relations of food to body-weight have been made by two methods; one of these is by estimating the amount of carbon and nitrogen excreted, on the theory that the need for repair is to be measured by the waste. The objection to this method is that a considerable proportion of what is regarded as the measure of wasted tissue may be imperfectly consumed excess. The other method is to measure the nitrogen and carbon in the food daily taken by different persons and classes. It will readily be seen that this plan also can lead to nothing better than an approximate result, inasmuch as we constantly see in some persons a fair average degree of health and capacity for exertion maintained by one half the quantity of food that seems to be required by others.

The investigations of the late Edward Smith, of London, upon this point, which were made for Government, were upon different classes of laborers, and showed that the adult body requires an average minimum daily amount of carbon of 9½ to 10½ ounces in the middle and light laboring classes, and of 12½ to 14 ounces in the ordinarily hard-laboring classes. Taking the relation of the weight of the body to the daily requirements of carbon, he showed that it was about 28 grains of carbon to each pound of body-weight; that is, supposing the weight of the individual to be 150 pounds, he would require 4,200 grains of carbon daily. In regard to nitrogen, the results of numerous investigations showed that about 200 grains of nitrogen are consumed daily by the light-laboring classes, while in the middle and well-fed classes it amounted to 260 grains. The relations which these amounts bore to body-weight varied from 1 to 1[illegible] grains to the pound. It should be remarked here that the nitrogen daily excreted does not depend upon the amount of muscular work performed, but mainly upon the amount ingested. The salts taken in the food are probably always in excess of the amount required, since the amount excreted depends upon the kind of food taken, as well as that resulting from the waste of the tissues.

The water required, is, of course, largely in excess of that which enters into the composition of the tissues, since it is the vehicle for the ingestion of the food and the excretion of the waste products. It also varies with temperature and barometrical pressure. It is estimated, however, that not less than five pints are necessary for an average adult at a moderate temperature and with ordinary exertion.*

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* Bence Jones, in his "Lectures on the Applications of Chemistry and Mechanics to Pathology and Therapeutics," makes the following estimate of the food required by the adult for daily nutrition under ordinary conditions: "Let us suppose that the minimum diet of health may be represented by 162 grains of soluble nitrogen, and 3,696 grains—nearly 8 ounces—of soluble carbon; then if 1 ounce of meat when cooked, contains 77 grains of nitrogen and 92 grains of carbon; or, if 1 egg contains 16 grains of nitrogen and 118 grains of carbon, and 1 ounce of butter contains 355 grains of carbon, and 5 ounces of dry bread contain 38 grains of nitrogen and 592 grains of carbon, and 21 ounces of arrowroot contain 3,633 grains of carbon, it is evident that

In considering, however, the amount of food required for the maintenance of the living and working body, I must impress upon you the fact which is too commonly ignored in the prescription of diet, viz., that the quantity is necessarily a variable one and must be proportioned to the age of the individual, and to the amount and nature of the work to be performed. Unless this is constantly borne in mind, the most serious consequences must ensue. In infants, for example, and during the whole period of body growth, the amount of food required, in proportion to the weight, is from three to five times greater than that required by the average-sized and ordinarily worked adult, provision having to be made for the growth as well as for the daily amount of energy to be expended in animal heat and mechanical work. In the adult, regard must constantly be paid to the protection enjoyed against heat and cold, and to the amount and kind of force expended in the occu-

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2 ounces of dry meat and 2 eggs, and 14 ounces of bread will give more nitrogen than the system requires to repair ordinary losses, and the excess may have to be thrown out, partly in the form of urates, when the oxidation is insufficient to produce urea.

"If 2 ounces of dry meat and 2 eggs only are taken, about 10 ounces of butter would be necessary to furnish the fuel and power for the system, or an equivalent quantity of fat must be absorbed from the stores deposited in the cellular tissue; or, if 5 ounces of dry bread only were eaten, then above 9 ounces of fat must be taken up in 24 hours; but a diet of 2 ounces of dry meat and 2 eggs, and 5 ounces of bread, requiring the wasting of the body by 7 or 8 ounces of fat daily, could not long be endured. Moreover, these quantities of nitrogenous food produce more urates than a less nitrogenous diet would do.

"If a little more than 21 ounces of dry arrowroot were taken daily, this would supply more carbon than is lost in the daily oxidation. The excess of carbon must either be deposited as fat, or must take away the oxygen, so as to leave none free to act on the nitrogenous substance that is passing out from the albuminous textures. Hence, with carbonaceous diet in excess, the whole of the uric acid from the tissues might pass off through the blood unoxidized."

pation. According to modern physiology, the source of all vital energy, whether in the form of heat, of muscular or nervous force, resides in the food, and the function of the animal body is to render active the potential energy which is stored up in the food we consume. "There is no motion without some equivalent antecedent motion;" * and as Dr. Frankland forcibly expresses it, "An animal, however high its organization may be, can no more *generate* an amount of force capable of moving a grain of sand, than a stone can fall upwards or a locomotive drive a train without fuel."†

I have entered thus briefly into the relations of the quantity of food required for daily nutrition, because it has a direct bearing upon the formation of the gouty habit. An excess of food is one of the most important factors in the production of the errors which result from sub-oxidation. This excess may be positive or relative; positive, in being more than can, by any possibility, be oxidized, and relative, in being more than is required by the habits and occupation of the individual. It is always to be borne in mind, therefore, in regulating the quantity of food necessary for healthy nutrition that it is to be proportioned to the age and to the forces which are to be provided for in the maintenance of animal heat and nervous and muscular energies. Any excess beyond these necessities being certain to escape thorough combustion, and lay the foundation of disease.

The relations of the quality of the food in the production of the gouty diathesis, though less important, probably, than those of quantity, are very striking and deserve special attention. It would appear, from the investigations of Edward Smith, Frankland, Haughton, and others, that the carbonaceous elements of the food are especially adapted to the production of heat and mechanical force, and that, with a free supply of oxygen, a diet of farinaceous, oleaginous and saccharine foods is best suited for persons requiring protection against cold, and engaged in occupations which demand great and prolonged muscular exertion. This corresponds with the well-known fact, that in the Arctic regions large quantities of fats are instinctively consumed, and that a diet containing a large proportion of carbonaceous substances is preferred by laborers. It is also in accordance with the diet regulations, founded upon experimental investigations, which are now frequently prescribed for the employees in factories, mines, and public works.

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* Bence Jones. Loc. cit.

† A lecture on the "Source of Muscular Power," by Edward Frankland, Prof. of Chemistry, Royal Institution, London.

A nitrogenous diet, on the other hand, with a minimum of carbonaceous aliments, seems to be better suited to the maintenance of a healthy nutrition in persons who do not require the evolution of a large amount of animal heat or whose occupations necessitate mental rather than muscular work. Clinical experience certainly shows that the characteristic dyspepsia of gouty persons is especially associated with the ingestion of large quantities of carbonaceous foods and beverages, where this diet is combined with lazy habits, indoor occupations, and intellectual pursuits.

It will doubtless occur to you that there is an apparent inconsistency here between the theory that lithæmia, which is the basis of the gouty vice, is caused by the defective disintegration of albuminous foods and the special intolerance in gouty subjects of an excess of carbonaceous foods. The most plausible, and probably the correct explanation of this inconsistency is, that the habits of those who suffer from lithæmia do not necessitate the use of much of the kind of food which is best adapted for the production of mechanical energy, and their supply of oxygen is insufficient for the thorough combustion of either the albuminous or carbonaceous foods. By withdrawing a portion of the latter, the oxidation of the former becomes more perfect.

This intolerance of non-nitrogenous food in excess in gouty persons whatever, may be its explanation, is a fact of which your clinical experience will, I am sure, soon furnish you abundant evidence.

The fact will present itself to you, as most marked, in a feebler capacity for the digestion, first, of sugar, and especially sugar in a state of fermentation, as we find it in cider, beer, and wine; second, of farinaceous foods, and third, of the fats. I have stated these substances in the order in which, I believe, experience generally teaches them to be productive of indigestion in gouty persons.

The effect of fermented liquors in the formation of the gouty habit scarcely needs mention, but in these days, when distilled spirits are made to bear the special blame of the physical as well as the moral evils of intemperance, it may not be out of place to venture the opinon that so far certainly as physical ailments are concerned, the ingestion of a given amount of alcohol in distilled spirits and in wine or beer, the former may be regarded as much less productive of mischief than the latter. It may be stated in this connection that the geographical distribution of the more severe manifestations of gout, shows that they are much more common in wine and beer drinking countries than in those where distilled spirits are chiefly consumed.

The tendency of sweet to provoke an acid and flatulent dyspepsia and the very special intolerance of them in persons in whom the gouty vice is well marked, is a common observation. This intolerance is especially observed in the children of gouty parents, and I mention it because sugar in all forms is so freely and indiscriminately given to children, in accordance with the popular notion that it is a most excellent food for them. I should also remark that fruits which contain a large percentage of glucose are among the articles of food which frequently require to be prohibited in the diet of gouty persons.

The inability to digest farinaceous substances in considerable quantities, observed in persons who inherit or who have acquired the gouty habit, demands special attention. The disposition to feed children indiscriminately upon large quantities of starchy food to the exclusion, oftentimes, of milk and other albuminous aliments, and to make them the staple of ordinary diet for adults, without regard to their habits and occupation, is another popular error, and is frequently persisted in, when it becomes the source of serious mischief. Of the starchy foods in common use, those generally preferred as most digestible and nutritious, are those which contain the largest percentage of starch, and the smallest percentage of the glutinous or albuminous principle. Thus rice, arrow-root, and potatoes are considered especially adapted for what is termed a light and nutritious diet, when they are, in truth, the most frequent source of indigestion in children and adults who live indoors and use little muscular exertion. If they do not cause indigestion they are transformed into fat, which is regarded as an indication of vigorous nutrition, when it is but too frequently the sign of an inherited or acquired tendency to that defective power in the thorough disintegration of food which leads sooner or later to disease.

The special derangements provoked by this intolerance of farinaceous foods in gouty persons, are disturbances in the primary digestion indicated by the signs of acid dyspepsia, acid urine and attendant gravel, and by the catarrhal affections of the mucous membranes and skin, which I have described to you.

There is generally less intolerance of the oleaginous aliments in gouty persons than of sweets and starchy foods. Cooked fats, and especially those which have been fried, are less apt to be digested easily than butter and salad oil. It is a fortunate circumstance that this ability to digest fats remains when sweets and farinaceous substances are not tolerated. In diabetes, for example, and in gout as well, the use of butter, cheese, and oil can be made to give a sufficiency of the carbonaceous elements to gratify the appetite and to subserve their special purposes in nutrition.

The second point in the treatment of the gouty diathesis to which I wish to draw your attention, is the necessity of a free supply of oxygen.

The diurnal amount of inspired air in the average adult varies, according to different observers, from 360 to 398 cubic feet. This, of course, is only an approximate estimate, as the amount must vary with all the circumstances, which affect the so-called vital capacity, and the rapidity of respiration. As I remarked in speaking of the causes of sub-oxidation, the loss of balance between the material to be oxidized and the air which oxidizes, is probably one of the most common causes of this defect. If much food is taken much air must be breathed. But it is not upon the quantity of air alone that thorough combustion of the food depends, the quality unquestionably affects its oxidizing power. It is not yet determined that anything is gained in the treatment of diseases depending upon sub-oxidation, by the administration of pure oxygen, or a less dilute mixture of it than atmospheric air, but it is very certain, that the highly heated and dry air of our furnace-warmed houses, is greatly inferior to the out-door air as an oxidizing agent. The colder the air the more oxygen will the lungs obtain in a given space, the hotter the air, the more diluted the oxygen.

Purity of air also doubtless affects its oxidizing power. If the proportion of carbónic acid be increased, or if other gases be present, the entrance of oxygen into the blood must be seriously obstructed the interference with diffusion in the air vesicles. The oxidizing power of the air is also supposed to be affected by the presence of ozone, and probably also by electrical conditions. It is through these agencies, perhaps, that the superior invigorating qualities of sea and mountain air are to be explained. We all know how a change of air will sharpen the appetite and quicken the process of disintegration, and it is not impossible that science may yet improve our means of treating the manifold disorders which arise from imperfect transformation of the food by suggesting artificial modifications of the respired air. For the present, however, we have to be contented with the oxygen as we find it in the atmosphere, and to make this efficient, we are compelled to inculcate the necessity of work or exercise in order to quicken the respiration and circulation, and thus secure its abundant supply and distribution.

I have thus sketched the relations which the quantity and quality of food and the free supply of oxygen bear to the treatment of the causes which determine the gouty vice. The next subject to which I shall ask your attention, is the medicinal management of this vice.

The principles involved in the treatment of the gouty diathesis, are the improvement of the oxidation of the food, and the promotion of the elimination of the effete products. The investigations of physiological chemists seem to indicate that the remedies upon which most reliance is to be placed in improving the process of oxidation in the blood are *Alkali* and *Iron.* Clinical experience, however, furnishes abundant evidence of the value of alkalies in the gouty habit. There is no class of medicines more universally applied for the varied manifestations of this vice, and none upon which such uniformly good results are obtained. The very fact that nature has provided them in such abundant proportion in the food we consume, would seem to indicate their necessity in the chemistry of nutrition. The reputation of mineral Spas also in the treatment of gouty affections bears universal testimony to their efficacy as medicinal agents.

Alkalies are used in medicine in the form of solutions of the caustic alkalies, lime, potash, soda, lithia, and ammonia, the alkaline carbonates, and the neutral salts of the vegetable acids, as the acetates, citrates, and tartrates. The latter are converted into carbonates before entering the circulation. The relative power of the alkalies in promoting oxidation within the body is not determined, though experience generally favors the use of soda as the least irritating to the stomach, and on this account best adapted for continuous use. Potash seems to have greater power as an eliminant, and is probably not inferior to soda in its effect upon oxidation.

Ammonia, in consequence of its volatility, probably has the least oxidizing power. Lithia has been highly lauded as a solvent of uric acid.

The time and mode of administration of alkalies are points of considerable importance. When they are required as means of assisting the disintegration of the food, they should be given with or soon after the meals; when they are needed to promote the elimination of effete matters, they act more efficiently when taken, largely diluted, upon an empty stomach.

The form in which these remedies are administered also deserves attention. They should always be given well diluted. This appears to be much more important than the quantity. The utility of mineral waters is not dependent probably so much upon the popular notion of nature's superior chemical combinations as upon the degree of dilution of the salts. In persons of gouty habit I usually prescribe Vichy water, which contains about forty grains of carbonate of soda to the pint, with the meals, and with most excellent effects.

Iron acts as an oxidizer by virtue of its increasing the red blood-globules, which are the carriers of oxygen. There are other metallic tonics which seem to act indirectly in the same way. Mercury, for example, in the spanæmia which occurs in the syphilitic dyscrasia, acts more efficiently in the same direction than iron; for most of the anæmic states, however, which arise from defective nutrition, and especially that which occurs in sufferers from chronic gout, there are no means for increasing the red globules and so improving the oxidation of the food equal to the preparations of iron. I do not propose to speak of the relative merits of the different forms in which iron is used, but would simply remark that the most efficient method, in my experience, of administering iron, appears to be that which produces the best results in the use of alkalies, viz.: largely diluted preparations of the soluble salts. The form in which iron is found in chalybeate waters combined with alkalies probably furnishes the best model for artificial preparations. The double salts of the vegetable acids, which are very soluble, largely diluted, constitute perhaps the most popular and the most efficient preparations of this remedy. The mineral acids are remedies of great value in the treatment of the gouty form of dyspepsia, but they appear to exert their good effects by virtue of their tonic properties and their influence in promoting primary digestion. As dialytic remedies, that is to say, as means of promoting oxidation, they are certainly inferior to the alkalies. They may sometimes be advantageously given at the same time, the alkalies being given on an empty stomach to exert their good effects in the circulation, and the acids immediately after the meal to promote the primary digestion.

To promote elimination your attention must be directed to securing a proper action of all the organs through which the waste products are discharged. The bowels, the liver; the kidneys, the skin, and the lungs must all be maintained in the most favorable condition for the discharge of their eliminative functions. The nitrogenous waste passes off mainly by the kidneys and bowels, and only under special morbid conditions by the skin. The skin throws off carbonic acid and certain volatile acids and salts, and the lungs, carbonic acid and water. I need not detain you with the details which are required to secure the healthy action of these organs. The use of purgatives for the occasional emergencies which necessitate prompt and efficient discharge of effete matter, of mild laxatives often requisite to promote the diurnal needs, the use of diuretics to stimulate the free discharge of the nitrogenous waste, the daily bathing and frictions to keep the skin clean and active, and the free exercise of the lungs to prevent the stagnating effects of accumulated carbonic acid, are all important details in the treatment of the morbid conditions arising out of imperfect oxidation, with which experience will soon make you familiar.

In conclusion I would remark that there are many details in the etiology, symptomatology, and treatment of the gouty vice, to which in future lectures I shall draw your attention. I have thus far confined myself to the enunciation of general principles, both as regards the causation and therapeutics of this constitutional malady. As you have doubtless observed, I have grouped a great variety of affections as belonging to this vice, but I trust I have succeeded in impressing you with the reasons which, it seems to me, justify us in referring this large and varied class of ailments to a special disturbance of the process of nutrition. There is certainly nothing strained in the idea, and there is much in clinical experience to confirm it, that, putting aside the diseases which arise from accident and parasitism, used in its largest sense, the majority of human ailments come from the violation of the laws which govern healthy nutrition; and where do we find the violation of these laws more frequently or forcibly illustrated than in the ignorance and intemperance which govern man in the use of food.

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